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THE ROLE OF PERIODIC EXAMINATION IN THE PREVENTION OF COALWORKERS' PNEUMOCONIOSIS

BY

A. L. COCHRANE, C. M. FLETCHER, J. C. GILSON, and P. HUGH-JONES

From the Pneumoconiosis Research Unit of the Medical Research Council, Llandough Hospital, Nr. Cardiff

There can be no doubt that coalworkers' pneumoconiosis presents one of the most serious problems in industrial medicine in Britain, at least in respect of the numbers of men affected. From 1931 up to the end of 1949 some 36,000 men had been officially diagnosed by the Silicosis Medical Board or the Pneumoconiosis Panels of the Ministry of National Insurance as being disabled by the disease. Over 80% of these cases had arisen in the South Wales coalfield. The social consequences of the disease in South Wales have been reviewed by Hugh-Jones and Fletcher (1950). The cost of the disease in terms of compensation has been enormous and in terms of human suffering immeasurable.

The disease is one that has been shown to be due to the inhalation of coal dust (Hart and Aslett, 1942) and its ultimate control and prevention will come from dust suppression. Airborne dust concentrations underground must be reduced to levels at which they will not cause pneumoconiosis in men exposed to them throughout a normal working life.

Coal dust in some mines appears to be less dangerous than in others. But since the factors which may make coal dust dangerous are not yet known, the precise levels of dust concentration in different pits which may be regarded as safe are uncertain, and indeed they cannot be established until a generation of coal miners has been exposed to them for their working life and has been shown to remain healthy. At present the Ministry of Fuel and Power and the National Coal Board have accepted as provisional levels for " approved dust conditions " under which men may be expected to work with reasonable safety, 650 particles/cc.* between 1 and 5 microns in anthracite pits and 850 particles/cc., within the same size range, in steam and bituminous coal pits. This expectation is, however, based on scanty evidence (Bedford and Warner, 1942), and the approved dust levels are really no more than a provisional target for engineers engaged on dust suppression.

Since the complete suppression of all airborne dust in the mines is impracticable and safe levels of dustiness are not yet known, we believe that the introduction of a system of periodic medical examination of coal miners would be the procedure which would most quickly and economically lead to the prevention of pneumoconiosis.

All coal miners in France and in New South Wales are given a periodic medical examination, and this system is being introduced into the coal mines of South Africa and Belgium. Since the value of such an examination has recently been questioned by Meiklejohn (1950) we review the advantages it could bring in British coal mines.

The Objects of Periodic Examination

A system of periodic examinations would have three main advantages.

First, it would provide an immediate protection to the men in the interval before safe dust levels are defined and effective dust suppression achieved. This would be possible because symptoms and disability develop late in the course of coalworkers' pneumoconiosis while radiological changes, which indicate a danger of ultimate disability, nearly always appear before functional injury has been done to the lungs. Permanent injury could be prevented in men showing these early radiological changes if they were advised to work under conditions of reduced dust exposure.*

Secondly, it would reveal the mines, seams or even districts, where early signs of the disease were still arising, in spite of dust suppression, so that the attention of the engineers could be concentrated

* Estimated by the thermal precipitator.

* It has been stated (Lehmann, 1938 ; Craw, 1947 ; Smith, 1947) that some men are more susceptible to the effects of dust than others. But the methods suggested for detecting these men are so imprecise that serious manpower wastage would occur if all doubtful cases were barred from employment. Even if such men exist, they would be detected by their earlier development of abnormality on serial *x*-ray examination.

at appropriate places. To suppress dust where the disease is not arising is an economic and technical waste since the cost of dust suppression rises steeply as more control is demanded ; the law of diminishing returns applies forcibly after the simpler methods of dust suppression have been installed. We have seen noticeable radiographic changes occurring in previously normal men after only two and a half years' exposure to dust concentrations somewhat in excess of " approved " conditions, so that radiological control should provide biological evidence of inadequate dust control within a period short enough for effective action to be taken.

Thirdly, if periodic examination were combined with routine dust studies at representative pits throughout the country, it should be possible in the course of time to determine the level of dustiness which had not given rise to any pneumoconiosis. In this way " safe " standards of dustiness could be defined and so permit adequate dust control by physical methods. It is conceivable that such an investigation might also throw light on the special features of the different dusts that are responsible for their varying pathogenicity, and so assist in predicting safe conditions for new mines.

The Basis of Periodic Examination

The last two objectives of periodic examination which we have outlined above, those leading to the maintenance of a safe working environment by dust suppression, require no further justification. The first, that of providing immediate protection for the men, depends on two pieces of medical evidence, namely (1) that early radiological changes can be detected before serious disablement occurs ; (2) that if men with these early radiological changes are no longer exposed to dangerous dust, they will not become disabled by the dust that they have already retained in their lungs. This evidence will therefore briefly be reviewed.

(1) Disability in Relation to Radiological Change.—There are two types of radiological change which may occur in coalworkers' pneumoconiosis : " simple pneumoconiosis " and " progressive massive fibrosis ", P.M.F., (Fletcher, 1948). There is evidence both from pathological (Gough, 1949 ; Heppleston, 1947) and radiological follow-up studies (Davies, Fletcher, Mann, and Stewart, 1949) to suggest that each represents a distinct pathological process. The combination of simple pneumoconiosis and progressive massive fibrosis has been called " complicated pneumoconiosis ".

A quantitative radiological classification (Fletcher, Mann, Davies, Cochrane, Gilson, and Hugh-Jones, 1949) defining four categories of increasing amounts of simple pneumoconiosis (categories 1–4) and four of progressive massive fibrosis (categories A–D) will be used throughout this paper.

Simple Pneumoconiosis.—Coal dust which is retained in the lungs gradually accumulates in small, scattered, discrete foci (Heppleston, 1949 ; Gough, James, and Wentworth, 1949) and this process is seen radiologically by the gradual appearance of characteristic minute opacities.

Progressive Massive Fibrosis.—This process is characterized pathologically by the development of larger nodules of collagenous fibrosis (called " infective " nodules by Gough) which are seen on the radiograph as massive shadows (" massive consolidation " of Sutherland and Bryson, 1929 ; " conglomerate shadows " of Bloomfield, Dallavalle, Jones, Dreessen, Brundage, and Britten, 1935 ; " images pseudo tumorales " of Eck and Hanaut, 1944 ; etc.).

The M.R.C. Pneumoconiosis Research Unit has recently investigated the relation between these radiological appearances of pneumoconiosis and the degree of respiratory disability in different age groups of South Wales coal miners, using clinical assessment and a variety of physiological tests.

The subjects were selected by their age and their radiological category of pneumoconiosis, and men were rejected from the experimental group only if they had a disease other than pneumoconiosis which might independently affect their respiratory function, e.g. hypertension, chest injury, etc. Normal subjects chosen as controls were men who had never worked in the coal mines ; the majority were attending the Ministry of Labour's employment exchanges between jobs. A full account of this investigation will be published elsewhere.

It was found that excessive breathlessness on effort, the main disability in pneumoconiosis, was not caused by a marked increase in the ventilatory requirements for a given amount of exercise, but by a decrease in the ventilatory reserve. Reduction in the maximum breathing capacity test (M.B.C.) was found to be the best simple index of this lowered reserve and to be closely related to the man's complaint of breathlessness on exercise. This is generally accepted (Böhme, 1938 ; Jequier-Doge and Lob, 1944 ; Rossier, Bucher and Wiesinger, 1947 ; Baldwin, Cournand, and Richards, 1948), and here we shall only consider the results obtained with this test. The test was done in the manner described by Gilson and Hugh-Jones (1949) and the results are given in Table 1.*

* D'Silva and Mendel (1950) showed that the M.B.C. measured with a water spirometer might be subject to considerable error. We have fully investigated the errors of the test in the circumstances in which our results were obtained ; they are small and can have no significant effect on the results reported.

TABLE 1

MEAN MAXIMUM BREATHING CAPACITY (LITRES/MIN.) IN THREE DIFFERENT AGE GROUPS OF MINERS AND OTHERS AT DIFFERENT RADIOLOGICAL STAGES OF PNEUMOCONIOSIS*

Age (years)	Men without Chest Disease	Miners with Less than Category 1 Simple Pneumoconiosis	Miners with Simple Pneumoconiosis		Miners with Progressive Massive Fibrosis	
			Early (Category 1 & 2)	Advanced (Category 3 & 4)	Moderate (Stage B & C)	Advanced (Stage D)
	×	○	◒	●	▭	■
35 (1·4)	145 (30·0) N = 10	119 (29·2) N = 18	115 (31·5) N = 29	135 (22·4) N = 8	98 (23·8) N = 8	65 (19·8) N = 6
45 (1·9)	131 (18·6) N = 10	109·5 (26·4) N = 19	110 (24·2) N = 27	101 (28·2) N = 8	79 (31·6) N = 8	51 (22·5) N = 8
55 (1·7)	118 (27·6) N = 10	84 (28·4) N = 31	97 (30·5) N = 30	87 (22·3) N = 16	61 (7·6) N = 8	33 (10·7) N = 8

* The standard deviation is given in parenthesis. N = number of subjects.

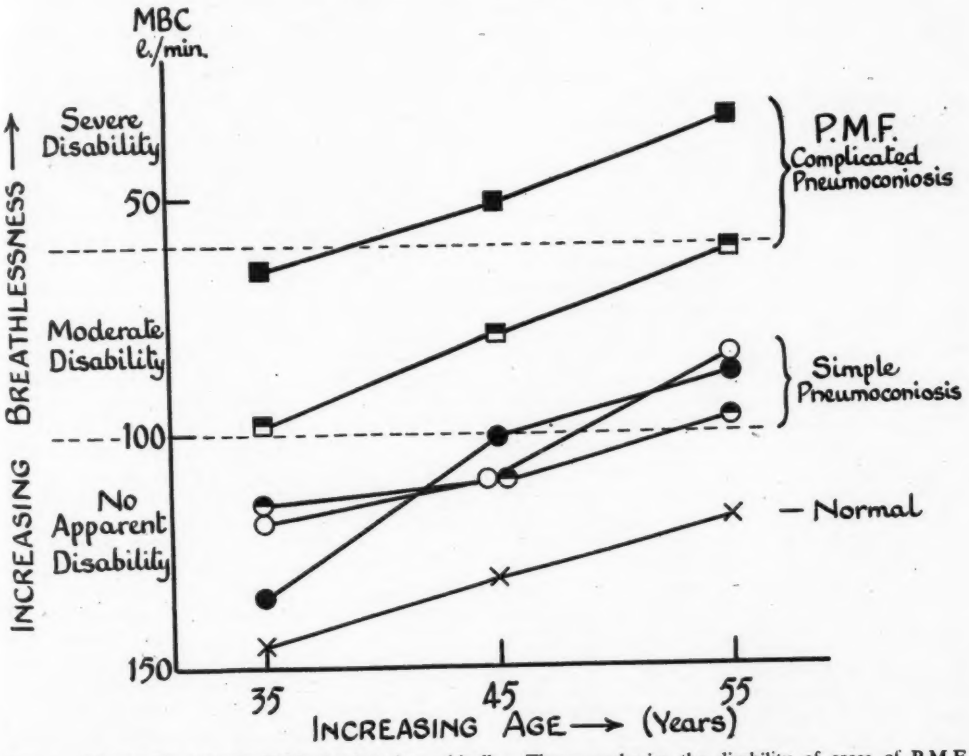

FIG. 1.—The results given in Table 1 plotted graphically. These emphasize the disability of cases of P.M.F. relative to age compared with that in cases of simple pneumoconiosis. (For meaning of symbols see Table 1.)

It will be seen from Table 1 that in each radiological group there is a decrease in the maximum breathing capacity with age, but in all age groups the maximum breathing capacity of the subjects who have progressive massive fibrosis is markedly reduced compared with those who only have simple pneumoconiosis. These findings are seen more clearly when the results in Table 1 are plotted graphically (Fig. 1).

From Fig. 1 it can be seen that cases of simple pneumoconiosis have on the average only a moderate systematic increase of the normal impairment of M.B.C. which occurs with age, and the radiological category of simple pneumoconiosis appears to have relatively little influence.* What is important for our purpose is that, on the average, young men even with quite advanced simple pneumoconiosis are not seriously disabled. Men with progressive massive fibrosis, on the other hand, are usually quite markedly disabled and their disability increases both with age and the radiological degree of the P.M.F.

Other workers have come to similar conclusions. Hart and Aslett (1942) found that coal miners with " reticulation " (the approximate equivalent of simple pneumoconiosis) only had a significantly greater degree of respiratory disability than miners with normal radiographs in the age groups 40–64, whereas in all age groups cases of " consolidation " (the equivalent of complicated pneumoconiosis) were considerably more disabled than men with either normal radiographs or with " reticulation ". Wright (1946), who considers respiratory disability to occur in coalworkers' pneumoconiosis only when " benign " anthracosis is complicated by silicosis, found that " simple discrete nodular silicosis is, with rare exceptions, entirely compatible with a working capacity adequate to allow full employment " but that " conglomerate shadows " are often seriously disabling. Bruce (1942) and Rossier, Bucher, and Wiesinger (1947) found that it was only in " silicosis stage III " (the equivalent of massive fibrosis) that any marked disability occurred.

There are, however, other workers who assert that the disability in pneumoconiosis bears no relation to the radiological appearances : for example, Peterson, Peterson, and Startup (1939) working with 36 South Wales coal miners ; Motley, Lang, and Gordon (1949) working with 100 American anthracite miners and later with 56 bituminous coal miners (Motley 1950). These opinions are based on results which are difficult to interpret since they were derived from samples which may have been biased towards more disabled men, and full allowance was not made for age. That this could explain anomalous findings is evident from Table 1 in which the wide divergence of individual results about the group means is given.

(2) The Progression of Pneumoconiosis after Cessation of Dust Exposure.—Our only evidence about the effects of pneumoconiosis once exposure to dangerous dust has ceased is from radiological studies.

Simple Pneumoconiosis.—Cases of simple pneumoconiosis can progress radiologically in two ways : the simple pneumoconiosis may increase as such, or progressive massive fibrosis may supervene. Davies and others (1949) showed that in the absence of coal dust inhalation no increase in the severity of the radiographic appearance of simple pneumoconiosis was seen over periods up to 10 years. Recently, in the course of follow-up studies of a Welsh steam-coal colliery, it was possible to obtain further evidence. Here 75% of the underground workers were examined by *x* rays in 1947 and again two and a half years later in 1949. The findings are given in Table 2. It will be seen that progression of simple pneumoconiosis only occurred in men who had been exposed to dust during the interval between the two surveys. No evidence is available on the progression of simple pneumoconiosis over longer periods than those covered in these two investigations.

The Onset of Progressive Massive Fibrosis.—Hart and Aslett (1942) reported that massive shadows in coalworkers' pneumoconiosis (the equivalent of P.M.F.) always appeared on a background of reticulation (the equivalent of simple pneumoconiosis) and we have confirmed this fact in all our studies of the disease.*

The question arises as to how much simple pneumoconiosis must be present before massive fibrosis may supervene and whether it can do so after the cessation of dust exposure.

Davies and others (1949) reported that 98% of cases of early massive fibrosis had a background of category 3 or 4 simple pneumoconiosis. This suggested that massive fibrosis only arose in cases with more than the lower limit of category 3 simple pneumoconiosis. If this were so, cases with category 1 or 2 simple pneumoconiosis should not be liable to massive fibrosis. Sometimes,

* It will be noted that the average M.B.C. of miners with less than radiological category 1 simple pneumoconiosis is less than the corresponding average for the normal in all age groups not engaged in coal mining. Much larger groups of men including " normal " subjects from different industries in South Wales, would be necessary to draw any firm conclusions from this finding. It was found, however, that the radiographs of those miners with minimal changes of pneumoconiosis could be distinguished from radiographs of non-miners by skilled observers.

* In cases of advanced massive fibrosis, concomitant bullous emphysema may obscure the simple pneumoconiosis. This may account for the occasional case of massive fibrosis without associated simple pneumoconiosis described by McVittie (1949).

TABLE 2

RADIOLOGICAL PROGRESSION OF SIMPLE PNEUMOCONIOSIS IN THE UNDERGROUND POPULATION OF A WELSH STEAM COAL COLLIERY DURING 2½ YEARS

Occupation during Interval	No. Showing Definite Progression* in 2½ Years	Total No. (%)
Coal face work ..	11	123 (8·9)
Stone work	7	97 (7·2)
Mixed stone and coal ..	2	50 (4·0)
Miscellaneous underground	4	79 (5·1)
Minimal exposure (haulage, etc.) ..	2	95 (2·1)
Outside mining ..	0	35 (0·0)
Dead or not followed ..	—	15 —
Total	26	494† (5·3)
Massive fibrosis obscuring the background of simple pneumoconiosis		5 —
Total		499 —

* "Definite progression" includes simple pneumoconiosis appearing in cases originally having a normal radiograph. † Thirty-three cases with early massive fibrosis, which did not interfere with the assessment of the underlying simple pneumoconiosis, are included.

however, it is difficult to read the radiological amount of simple pneumoconiosis when massive fibrosis has supervened and there is also the possibility that the simple pneumoconiosis may have progressed while the massive fibrosis was appearing. It is the category of the simple pneumoconiosis at the time of onset of the massive fibrosis that we really need to know.

Evidence of this nature was obtained during the follow-up study of the Welsh steam-coal colliery already described. In this study progressive massive fibrosis did not develop in any case with category 1 simple pneumoconiosis, but it did develop in some cases with category 2 as well as in those with category 3 simple pneumoconiosis; moreover, the attack rate was no less in category 2 than in category 3 cases (Table 3).

It appears that as simple pneumoconiosis progresses, there is an increasing liability to develop P.M.F. and that there is a relatively rapid increase in this liability within the stage of simple pneumoconiosis covered by category 2. This led Fletcher (1950) to suggest that there was a "critical stage" of simple pneumoconiosis at which liability to P.M.F. arose. The range of simple pneumoconiosis over which a significant risk of P.M.F. arises may

TABLE 3

ATTACK RATE* OF EARLY PROGRESSIVE MASSIVE FIBROSIS IN THE SAME POPULATION AS IN TABLE 2

Initial *X*-ray Category	Dead or Untraced	Number Developing Early P.M.F.	Total No.	Annual Attack Rate (%)
N	6	0	170	0
1	1	0	124	0
2	4	6	95	2·5
3	2	4	72	2·2
Total ..	13	10	461	0·9
Number who already had P.M.F. in 1947			37	—
Total			499	—

* Annual attack rate is the percentage of cases developing P.M.F per year.

vary in different parts of the country, and its exact definition must await the examination of larger numbers of cases. This could be done in the pilot scheme we propose below.

Davies and others (1949) also gave evidence to show that once a man has the requisite amount of simple pneumoconiosis his chance of developing massive fibrosis was independent of further dust exposure, and that massive fibrosis was nearly always progressive. This independence of dust exposure and attack rate of progressive massive fibrosis was again found in our recent follow-up study (Table 4).

We may summarize these radiological observations concerning the onset of massive fibrosis by saying that (1) if a man is removed from further exposure to dangerous dust when he has only a small amount of simple pneumoconiosis, he is most unlikely to develop progressive massive fibrosis; (2) once a man has enough simple pneumoconiosis, progressive massive fibrosis may develop with equal likelihood whether or not dust exposure ceases; (3) once massive fibrosis has started it is nearly always progressive, though at widely varying rates. We must emphasize that these conclusions only concern coalworkers' pneumoconiosis and are not necessarily applicable to silicosis.

The Significance of the Radiological Findings.—The importance of these findings is that they suggest how men might be protected from disabling pneumoconiosis by periodic *x*-ray examination. If men are prevented from developing more than early simple pneumoconiosis we know that they will not develop progressive massive fibrosis with its

TABLE 4

ANNUAL ATTACK RATE OF PROGRESSIVE MASSIVE FIBROSIS IN CASES OF SIMPLE PNEUMOCONIOSIS (CATEGORIES 2 AND 3 FROM TABLE 3) ANALYSED BY OCCUPATION DURING THE INTERVAL

Occupation	Total No.	No. Attacked by P.M.F.	Annual Attack Rate of P.M.F.
Coal face work	49	3	2·4
Stone work	19	2	4·2
Mixed stone and coal ..	17	1	2·4
Miscellaneous	42	2	1·9
Minimal (haulage, etc.)	21	1	1·9
Outside mining ..	13	1	3·1
Dead or untraced ..	6	—	—
Total	167	10	2·4

attendant disability. We can presume that they will not become significantly disabled by a small degree of simple pneumoconiosis (Table 1 and Fig. 1). An essential point in our argument is that since the early stages of simple pneumoconiosis are asymptomatic they can only be detected with certainty, and the men given suitable advice, by a system of periodic *x*-ray examination.

Practical Considerations

Advice after Examination.—From the foregoing discussion we can consider the advice that men should receive after their examination so that the greatest amount of protection may be given with the least possible interference with coal production. The advice would depend upon radiological appearances and age, though disability and economic factors would also have to be considered. According to their radiological appearances, the men would be grouped into two classes: (1) new entrants, men without pneumoconiosis, and men with only the very earliest radiological signs of pneumoconiosis. These men could from the start of the scheme obtain freedom from the risk of serious pneumoconiosis. They would be advised to change to a less dusty working environment if, on serial examination, they began to show evidence of simple pneumoconiosis whose severity was approaching the level at which a significant risk of massive fibrosis arises. The most important factor in giving this advice would be the rate of development of simple pneumoconiosis from one examination to the next. Age and industrial history would also have to be taken into account: a man of 55 who has early simple pneumoconiosis and has worked in the mines all his life is clearly unlikely to progress significantly in the rest of his working life. On the other hand, a man of 25 or 30 with the same radiological appearance may be running a serious risk. (2) Men who were found to have advanced simple pneumoconiosis or who had already contracted P.M.F. would not derive much benefit from periodic examination. They are the victims of the past. In view of the present evidence that the dust exposure has little effect on their liability towards progression they should as a rule not be advised to change their working environment.* The only basis for advising a man in this second group to leave the industry would be on account of disability, for it is clearly futile to advise a man to continue at a job of which he is already physically incapable. Moreover, certain cases, which on re-examination showed rapid radiological progression, might well be advised to leave mining, for it could confidently be forecast that they would become seriously disabled in the future and when they did so they might well blame this on having continued work. A small number of such cases might bring the system of advice into disrepute.

Obviously, in individual cases, there might be exceptions to the advice we have suggested. For example, if a young man were showing early radiological changes and less dusty working conditions were not available, he might still be advised temporarily to hold his job within the industry in areas such as South Wales, where there are slender chances of obtaining immediate alternative employment. The disease develops very slowly and there is no hurry for a man to leave the industry while seeking another job.

The phrase "less dusty working conditions" needs some comment. Men who are showing progression of simple pneumoconiosis in spite of exposure to relatively low dust conditions should be removed from any further dust exposure—by leaving the industry or taking up surface work. On the other hand, if the progression could be attributed to working in high dust concentrations, change to a less dusty, even if not dust-free environment, might be sufficient to arrest progression. It is thus apparent that in order to give appropriate advice doctors would have to have full information concerning the dust concentrations under which the

* In the course of periodic examination knowledge of the precise relationship between the attack rate of P.M.F. and the severity of simple pneumoconiosis would increase and some men who already had fairly advanced simple pneumoconiosis might come to be classified with the first group of early cases. This is especially true in coalfields outside South Wales where our knowledge of the relation between the degree of simple pneumoconiosis and chance of contracting P.M.F. is slight.

men had been working since the last examination, and concerning the concentrations prevailing at the alternative, less dusty working places that the N.C.B. could offer to them. There is real need for some form of continuous dust-sampler which could be used in assessing dust levels under " approved conditions ". This, used in conjunction with periodic examination, would show whether these conditions are, in fact, safe.

We may summarize by saying that the only men who would have to be advised to leave the industry or to change their occupation to one involving less dust exposure would be those with early simple pneumoconiosis which was found to be advancing on serial *x*-ray examination, especially if they were young, and those with more advanced radiological changes who were becoming disabled by the disease.

Compensation.—The purpose of the scheme is the prevention of disability. This should be made quite clear to the men by making it independent from any question of compensation. If the scheme were also concerned with compensation the emphasis would be removed from fit men whose health must be preserved to disabled men to whom little but financial compensation could be offered.

Men with pneumoconiosis who claimed disability could be referred to the Pneumoconiosis Medical Panels for disability assessment. *X*-ray examination under the periodic system could achieve an enormous economy by replacing the scrutiny examination at present carried out by the Pneumoconiosis Panel whose duties would then be confined to their statutory function of assessing disability and its cause. Arrangements would have to be made for the radiographs taken in the course of periodic examination to be lent to the Pneumoconiosis Panel.

If compensation issues were kept separate in this way, clinical examination could be confined to special cases. Radiological examination alone should suffice to detect the earliest stages of pneumoconiosis and to follow its progression towards dangerous levels.

Meiklejohn (1950) has objected that " the worker and his union will want to know whether he has contracted pneumoconiosis, not if he has reached the critical level of severity ". It is difficult to understand when a man can be said to have " contracted pneumoconiosis " since simple pneumoconiosis develops from normality by a gradual and continuous process. Experience has shown that miners are perfectly willing to accept the idea that there may be some evidence of dust in their lungs without there being any disease, by simple analogy with the fact that they commonly have dust in the " blue scars " on their skin without their having skin disease. The men's right to apply to the Pneumoconiosis Panel for compensation for disability would, of course, remain.

One alteration in the present system of compensation would be necessary: under the National Insurance (Industrial Injuries) 1946 Act no provision is made for giving compensation to men who, as a result of developing an early stage of an industrial disease, have to be advised to change their occupation although they are not disabled. Thus young men who would have to be advised to leave the coal mining industry under a system of periodic examination could at present receive no financial compensation. This defect in the provision of compensation would require rectification, possibly by legislation, if the scheme of periodic examination were to be a success.

The Introduction of the System.—In the organization of a scheme, such as we have outlined, there would be many administrative and technical problems whose importance cannot be forecast and whose solution could not be suggested in advance of the introduction of the scheme. For instance, we do not know the prevalence of unrecognized pneumoconiosis in various coalmining areas, which would largely determine the appropriate interval between examinations; what effect the examinations would have on the number of applications for disability benefit to the Pneumoconiosis Panel; what staff (medical, technical, and administrative) would comprise an efficient unit, and what the operational costs of such a unit would be; or how far and in what way arrangements would be made by the N.C.B. for young men with early pneumoconiosis to continue working in the mines under conditions of minimal dust exposure. Because of all these uncertainties it is our view that periodic examinations should first be introduced on a pilot basis.

Since new entrants and young men would derive the greatest benefit from examinations it might be suggested that the system should in the first place be confined to the younger workers or even to new entrants to the industry. Periodic examinations would have little to offer to men who had already reached an age at which further serious progression would be unlikely to occur before the normal retiring age or to men who already had relatively advanced pneumoconiosis. However, it is our belief that it would in fact be administratively simpler and psychologically better to extend the examinations to include all underground workers in whatever pits were chosen for introducing the scheme.

The next question is whether all underground

workers could be examined unless the system were made compulsory. In underground surveys carried out on a voluntary basis by the Pneumoconiosis Research Unit's mobile unit at six different collieries, four in South Wales and two elsewhere in Britain, over 99% of the workers have been examined. This high percentage was achieved by obtaining the full cooperation of the trade unions, by active propaganda and, by offering examinations and subsequent interviews fro.n 10 a.m. to 11 p.m. Routine examinations could not be made on this basis, but if the N.C.B. gave men time off without loss of pay in order to attend for examination in a mobile *x*-ray unit, there should be no difficulty in obtaining practically universal examination without any form of compulsion.

The main difficulty would be to decide where the pilot scheme should be started. Since in South Wales the Pneumoconiosis Panels are already being used by the men as an irregular system of periodic examination, the scheme might well start there. Later, when any preliminary difficulties had been overcome, the scheme should start, on a voluntary basis, in mines representative of various areas in the different coalfields; it may be that some of our conclusions are not applicable to pneumoconiosis arising in other coalfields, particularly in respect to the relationship between severity of simple pneumoconiosis and liability to massive fibrosis. Such gaps in our knowledge could be filled in the course of a few years by the pilot scheme provided that it was so designed that the records and results obtained from it could be used for research purposes. This would entail adequate provision for following-up men who leave the industry or move from one mine to another in the various coalfields. Many of the doubts and fears expressed by Meiklejohn (1950) would be resolved by a scheme introduced in this way.

Interval between Examinations.—The interval between examinations would vary from one part of the country to another but the pilot scheme would enable satisfactory intervals to be determined. In mines where the dust conditions were not satisfactory and where new cases of disease were appearing, it would have to be of the order of a year. In some parts of the country it would be unnecessary to have the examinations at more frequent intervals than three or four years unless practical experience showed that it was impossible to keep track of men examined, for little change in the extent or prevalence of the disease could be determined in a shorter interval than this.

Nature of the Examination.—We have indicated that the basis of the examination need only be radiological. At present no miniature radiographic technique is adequate for the early diagnosis of simple pneumoconiosis (Fletcher, 1951). It would therefore be necessary to use full size films. The M.R.C. mobile unit is capable of taking 20 full size radiographs per hour and processing them to the finished dry stage. This would probably be a sufficient speed of work for any field survey. Each mobile unit could work a 35-hour week: a seven-hour day for five days. Allowing 17 weeks a year for movement from place to place and for holidays, they would work 35 weeks in the year. At this rate each unit could take 24,500 films per year and 10 such units could easily examine the 700,000 men of the country with an average interval of two and a half to three years between examinations. This should be quite adequate.

The question still arises whether even large films would enable accurate diagnosis of early simple pneumoconiosis to be made. In view of the work reported by Fletcher and Oldham (1949) on the inconsistencies of radiological diagnosis, it would be necessary for the doctors running the scheme to pass some test of their ability to read films of early simple pneumoconiosis accurately and consistently.

Errors will occur even with the most skilled reader. For this reason Meiklejohn (1950) suggests that it might not "be prudent at this stage to proceed, recognizing as we must that the decisions involve human welfare and happiness". There are two possible errors of diagnosis which might cause human unhappiness. The first would be the failure to detect pneumoconiosis in a man approaching the critical stage who might thereafter progress to serious disability; he would, however, be no worse off after having had his periodic examination. The only harm therefore that could be done would be by the diagnosis of pneumoconiosis in a man who was not in fact affected by pneumoconiosis and might thus be unnecessarily alarmed and led to change his occupation. We feel that errors of this kind should be so infrequent as to be negligible. Such errors chiefly arise from films of poor technical quality, and these might be reduced if it were decided that every man with early pneumoconiosis should have further confirmatory films taken before being advised to change his job.

The Effect on Manpower.—Meiklejohn (1950) has expressed the fear that a scheme of this kind would reveal so much pneumoconiosis that large numbers of men would have to be advised to leave the industry and even if they were not so advised, there would be a danger of producing as many cases of neurosis as the number of cases of pneumoconiosis

that would be prevented. It has been the experience of the Pneumoconiosis Research Unit that their surveys have produced no such neurosis.

There is ample evidence that many men are greatly reassured after they have had a careful radiological examination and a personal interview with a doctor, to know that dust has not affected their lungs. It is not our view that periodic examinations would cause neurosis or that they would reduce manpower. Fear of the effects of dust can be a bar to recruitment (" Social Survey ", 1947). Periodic examinations should dispel this fear and thus assist recruitment. Even where fear of dust has not yet arisen, the examinations could be used to give practical demonstration to potential recruits that the community is interested in the health of its miners. There is ample testimony from other industries that medical supervision, coupled with engineering control of health hazards, is essential for the maintenance of manpower. Whether or not numbers of men would have to be advised to leave the industry because of the fact that they showed progressive simple pneumoconiosis in serial examination would depend on the effectiveness of the dust suppression now being carried out by the N.C.B. The doctor's role is to ensure that miners who are in danger of disablement by pneumoconiosis are protected, if necessary by advising them to leave the industry. It would be for the engineers to ensure that the dust levels were such that very few men would have to be so advised. Manpower is a major problem in the coal industry. A system which protected the young miner from injury to his health by this serious industrial hazard would reap a great dividend not only in human contentment but also in productivity.

Summary

A system of periodic *x*-ray examination of coal miners is suggested which should ensure the prevention of disabling pneumoconiosis.

Evidence is presented which shows that early radiological changes can be detected before serious disablement occurs, and that if men with these early changes are removed from exposure to dangerous dust they will then not develop the progressive massive fibrosis which is the cause of serious disability.

A pilot scheme would be necessary. Practical details are discussed, such as the advice to be given after the examination, the separation of the scheme from problems of compensation, the interval between radiographs, and the effect on manpower.

The periodic examination would provide a means of defining safe dust levels in different mines leading to control of the disease by economical dust suppression, meanwhile giving protection to new entrants and to workers not already affected.

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THE ACTION OF VARIABLE AMOUNTS OF QUARTZ ON THE LUNGS OF RATS

THE EXTENT OF PATHOLOGICAL CHANGE IN RELATION TO THE AMOUNT INJECTED

BY

S. C. RAY, E. J. KING, and C. V. HARRISON

From the Postgraduate Medical School, London

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It is generally agreed that once a foreign body has come to rest in the lung alveoli the chief mechanism of its elimination is the lymphatic system. Foreign bodies are first engulfed by the phagocytes and these migrate along the walls of the air spaces until they reach the nearest lymph vessels, from which they ultimately reach the tracheo-bronchial lymph nodes. Therefore, small amounts of quartz which the lung can eliminate through its lymphatic system into the lymph nodes should not produce silicotic lesions in the lung parenchyma, but might cause them in the nodes, where most of the silica will ultimately be trapped.

Larger quantities of quartz, however, might be expected to overload the lymphatic drainage system, and, being retained in the lung, produce silicosis there. To test the truth of this assertion we have administered 2 mg., 5 mg., 10 mg., 30 mg. and 75 mg. doses of quartz of small particle size by intratracheal injection.

The sample of quartz used was from a commercial source.* On analysis it was found to contain 97·1% of SiO_2. (The balance consisted of H_2O, Ca, Fe, and Mg carbonates.)

The size distribution of the particles, determined by the method of Nagelschmidt (1943), is shown in Table 1.

The silica solubility was determined by the procedure described by King (1945, 1947), and was found to be 3·4 mg. SiO_2/100 ml. (buffered Ringer solution (*p*H 7·4) for 48 hours at 37°C.).

TABLE 1

SIZE DISTRIBUTION OF POWDERED QUARTZ

Size (μ)	Percentage of Quartz Particles	
	By Number	By Mass
Under 0·25	7·7	0·04
„ 0·5	28·9	0·8
„ 1·0	67·1	12
„ 1·5	83·6	20
„ 2	91·9	31
„ 3	98·0	54
„ 4	99·0	62
„ 5	99·5	70
„ 6·8	100·0	100

Method

The rats used were the black and white piebald variety (Medical Research Council strain). For each group the appropriate dose of quartz was suspended in a standard volume of 1·5 ml. because this has been found to be well tolerated by rats of 150 to 200 g. weight. Milk, 3%, was added to help to hold the dust in suspension. The suspensions were sterilized by heating in a boiling-water bath for about 10 minutes.

The rats were anaesthetized lightly, and the trachea was exposed by dissection. The 1·5 ml. of quartz suspension was then injected intratracheally, using Kettle and Hilton's (1932) method. During the procedure both the flask and the syringe into which the suspensions were drawn were kept agitated to prevent sedimentation. The injection was done quickly and with a certain amount of force to disperse the dust well into the lung alveoli. The wound was then closed by a single suture. There was no death due to the operation.

Pathological Technique

The lungs of the rats that died or were killed were removed after insufflation to normal size with 10% formol saline. Fixation, imbedding, sectioning, and histological examination were carried out according to the technique of Belt and King (1945). Due to cannibalism and untimely deaths, it was not possible to obtain exactly equal days of survival in the several experiments. But, as far as has been possible, sufficient intervals have been maintained between killings to study the progress of the lesions on a comparative basis.

* Messrs. Colin Stewart, Ltd.

In assessing the degree of pathological change, four grades of fibrosis have been recognized : grade 1, loose reticulin fibrils with no collagen ; grade 2, compact reticulin with or without a little collagen ; grade 3, somewhat cellular but made up mostly of collagen ; grade 4, wholly composed of collagen fibres and completely acellular ; and grade 5, acellular, collagenous, and confluent. The details of the individual experiments are set out in Table 2.

TABLE 2

ASSESSMENT OF FIBROSIS AND AVERAGE MAXIMUM SIZE OF NODULES IN SECTIONS OF LUNGS OF RATS RECEIVING QUARTZ

Days of Survival	Size of Nodules (μ)	Grade of Fibrosis
	2 mg. Quartz	
22	—	0
50	0	0
77	0	0
103	0	0
105	0	0+ Fibrosis in lymph node
183	0	0 Some degree of peri-
293	0	0 vascular and peri-
295	0	0 bronchial fibrosis, and
365	0	0 fibrosis in lymph nodes
	5 mg. Quartz	
14	0	0
15	0	0
134	186	1 (minimal)
168	186	1
182	372	2
331	362	3
407	372	2
	10 mg. Quartz	
7	0	0
27	0	0
30	0	0
101	141	1 (minimal)
123	160	2
154	186	2
162	223	2
165	234	2
316	316	2
	30 mg. Quartz	
66	112	1 (minimal)
94	234	1
116	272	2
164	502	3
283	458	4
350	577	4
351	564	4
	75 mg. Quartz	
68	260	1
121	551	3
131	595	4
207	558	5
240	949	5
302	1135	5

Pathological Findings

Quartz 2 mg.—This amount did not produce any lesion until 105 days when some fibrosis was seen in the tracheo-bronchial lymph nodes. No fibrosis was seen in the lung parenchyma of rats killed, for example, at 183 days, but there was some perivascular and peribronchial fibrosis. These changes were seen again in rats killed after 295 days, and there was some fine reticulinosis of isolated alveoli, but this was too mild to be included under grade 1 fibrosis.

Microincineration followed by hydrochloric acid proved unsatisfactory for finding the very small amount of quartz present in these lungs. However, it was possible to recognize quartz by dark ground examination of the sections stained with haematoxylin and eosin and incinerated sections which had not been treated with hydrochloric acid. Using these methods quartz could be found in the lungs from the second to the 365th day ; but it was not possible to tell, except very roughly, whether the amount diminished during the course of the experiment. The impression was gained that there was more in the early sections than in the later, but this impression cannot be considered quantitatively.

Quartz 5 mg.—Dust cell reaction was marked in rats killed at 14 and 15 days, but there was no reticulinosis. After 134 days isolated foci of reticulinosis were seen with grade 1 (minimal) fibrosis. A rat killed after 168 days showed typical grade 1 fibrosis (Fig. 1). On the 182nd day nodules with grade 2 fibrosis were seen. One rat that died on the 331st day had well developed nodules with grade 3 fibrosis. But the rat that lived to 407 days showed only grade 2 fibrosis. In this and subsequent groups the lymph nodes were always involved when there was fibrosis of the lungs.

Quartz 10 mg.—No fibrosis was seen in the lungs of rats killed or dying in the first 30 days, although dust cell reactions remained well marked. On the 101st day areas of reticulinosis with grade 1 (minimal) fibrosis were present. Nodules with grade 2 fibrosis were seen in the lungs of rats after the 123rd day. This grade 2 fibrosis continued till 316 days (Fig. 2).

Quartz 30 mg.—Grade 1 fibrosis was present in the lungs of rats killed on the 66th and 94th days. On the 116th day the character had changed to grade 2, and on the 164th day to grade 3. The lungs of rats examined on or after 283 days showed grade 4 fibrosis (Fig. 3).

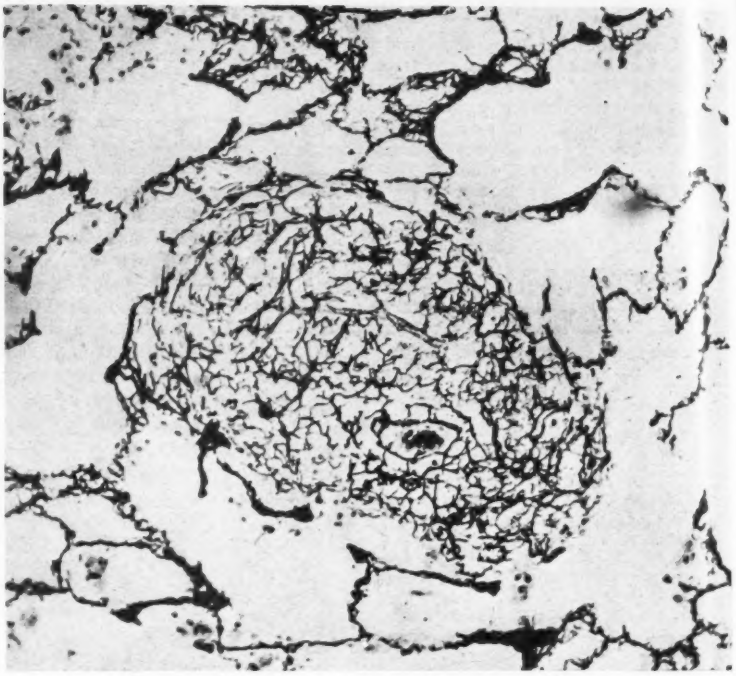

FIG. 1.—Rat lung, 168 days after a dose of 5 mg. of quartz, showing grade 1 fibrosis. There is a localized lesion consisting of a loose network of fine reticulin fibrils without any collagen. (Silver impregnation × 190).
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FIG. 2.—Rat ung, 316 days after a dose of 10 mg. of quartz, showing grade 2 fibrosis. The lesion contains a dense network of coarse reticulin fibrils, some of which are thick enough to give the staining reactions of collagen. (Silver impregnation × 190).
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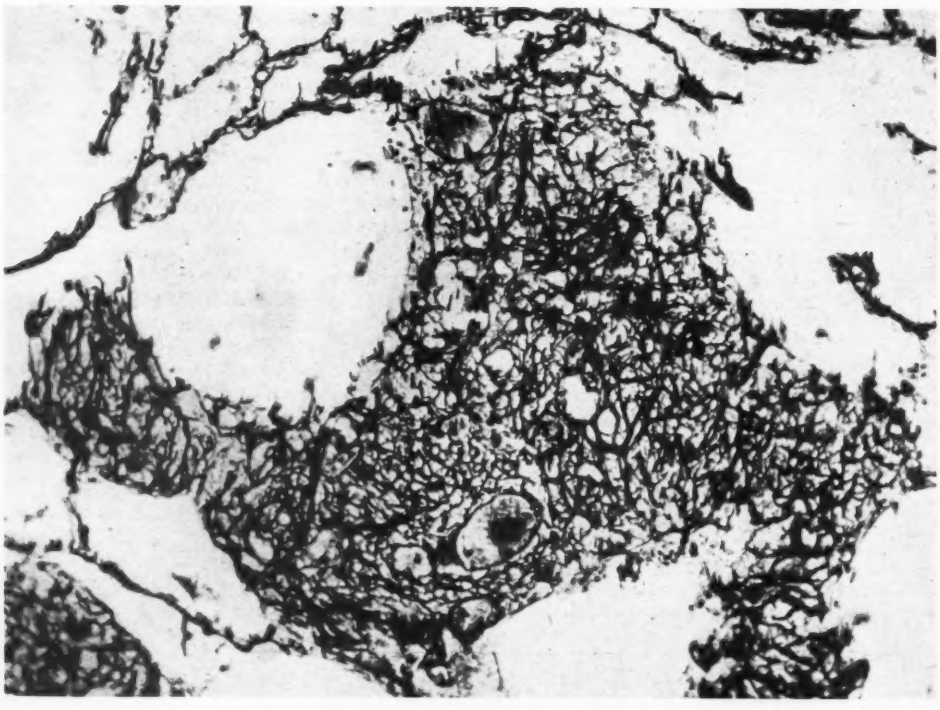

Quartz 75 mg.—A rat that was killed on the 68th day showed grade 1 fibrosis. Lungs of rats examined on the 121st day showed grade 3 fibrosis. On the 131st day the lungs had acellular and collagenous nodules of grade 4 fibrosis. This changed to grade 5 from 207 days onwards (Fig. 4).

To summarize : 2 mg. of quartz did not produce any lesion in the lung, but caused fibrosis of the nodes. On the other hand 5 mg. of quartz produced grade 1 (minimal) fibrosis in 134 days, as opposed to 101 days with 10 mg. of quartz. Grade 2 fibrosis was reached with 5 and 10 mg. of quartz in 182 and 123 days respectively ; and in both experiments this grade 2 fibrosis was not exceeded during 300 days (except one grade 3 with 5 mg. at 331 days). Grade 1 fibrosis was produced in 66 and 68 days by 30 mg. and 75 mg. of quartz, but with 30 mg. the fibrosis was minimal. The 30 and 75 mg. of quartz produced grade 3 fibrosis in 164 days and 121 days, and grade 4 and 5 in 283 and 207 days respectively. In both these cases the grade remained constant after 300 days, although with the higher concentration (75 mg.) the nodules became more confluent and massive in character.

Although the highest grade of fibrosis seen in the lungs has been mentioned, it should be pointed out that in animals receiving the larger amounts of quartz the lung sections after 300 days showed all grades of nodules.

To help to assess the pathogenic action of the various amounts of quartz quantitatively, the diameters of fully matured nodules were measured in each case. The results, with the grades of fibrosis, are given in Table 2. The final figure of the size of the nodules was obtained by measuring the diameters of the three largest nodules in two directions at right angles to each other and then taking the mean average figure of the three. The measurement was made with the help of a micro-millimetre scale. Photomicrographs of sections illustrating typical findings with the different amounts of quartz are given in the figures.

Discussion

These experiments are a repetition and extension of preliminary work already described (King, Harrison, and Ray, 1949), where 5 mg. of quartz appeared to be at least partly cleared from the lungs in many of the animals used, and even 10 mg. in some, and consequently produced little or no fibrosis. Because the results were not entirely uniform, the present experiments were planned on the basis of a wider range of quartz dosage. They well illustrate the disturbance of the mechanism of the physiological disposal of foreign matter from the lungs and the stages of development of animal silicosis. It is probable that, in the sequence of events, impairment of the drainage system of the lung constitutes the first stage in the development of silicosis.

Small amounts of quartz, namely 2 mg., are easily cleared from the lungs by the phagocytes, but are ultimately trapped in the tracheo-bronchial lymph nodes, where the progress of fibrosis is more rapid than in the lung parenchyma. With small amounts of quartz, therefore, fibrotic nodules first appear in the lymph nodes. The first irritative and proliferative reactions in the lung proper are seen round the vessels and bronchi, which are rich in lymph vessels, as was evident with 2 mg. of quartz at 183 days.

Larger amounts of quartz upset the mechanism of dust dispersal by impairing the lymph movement, and ultimately lymph stasis sets in within the lung itself. This gives a better chance for the accumulation of particles in the lung. With still larger amounts of quartz the whole cleaning mechanism breaks down, and more silica remains in the lung and thereby sets up inflammatory changes with the formation of dust granulomas and connective tissue proliferation in the lung tissue. This is the beginning of true silicosis, and is characterized by discrete nodules distributed throughout the lung.

The rate and severity with which quartz acts varies with the amount. With quartz of the same particle size, the larger the amount injected the greater is the area of lung tissue involved, and so also the nature or grade of fibrosis, when the same time factor is taken into consideration. The maximum size of nodules produced by 2, 5, 10, 30, and 75 mg. of quartz over 300 days are 0μ, 372μ, 316μ, 577μ, and 1135μ respectively.

The grades of fibrosis are 0, 2, 2, 4, and 5 respectively. (With 5 mg. of quartz grade 3 fibrosis was seen in one animal at 362 days ; this was probably due to a high concentration of quartz at one place, caused by unequal dispersal of the dust in the lung tissue.) With 5 and 10 mg. of quartz the difference is not so marked as with higher doses where it is quite obvious. It appears, therefore, that the tissue reactions in animals are more or less proportional to the concentration of quartz in the lung.

Summary

Small amounts of quartz (2 mg. in this experiment with rats) are mostly cleared from the lung and produce fibrosis in the lymph nodes only. With larger amounts the lymphatic drainage is insufficient to remove the dust from the lung, where it remains to produce its characteristic reaction.

The pathological response of the lungs to quartz varies with the amount : with 2 mg. no fibrosis was

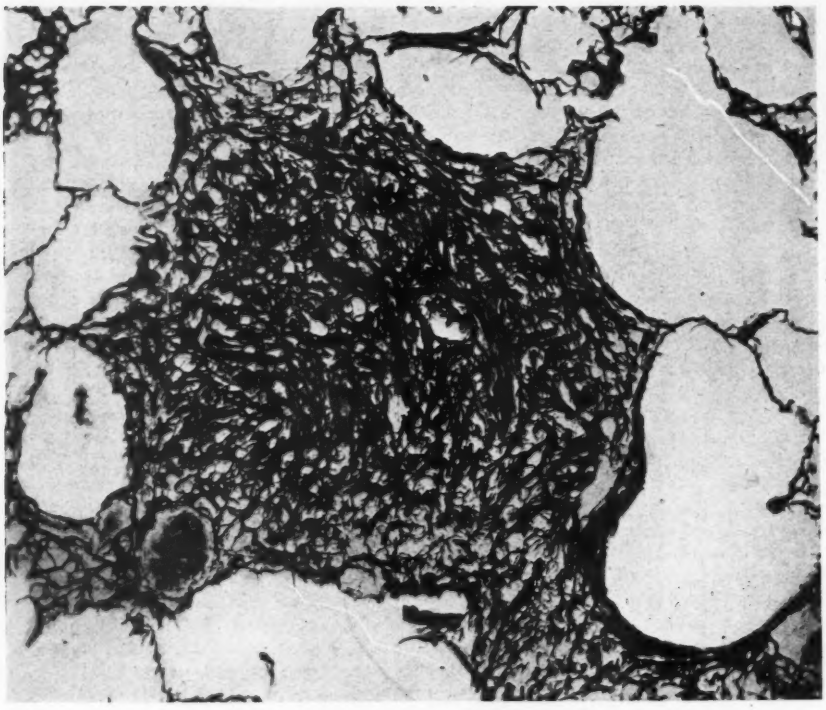

FIG. 3.—Rat lung, 350 days after a dose of 30 mg. of quartz, showing grade 4 fibrosis. The nodule consists of tangled collagen fibres, and with ordinary nuclear stains was practically acellular. (Silver impregnation × 190).

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FIG. 4.—Rat lung, 302 days after a dose of 75 mg. of quartz, showing grade 5 fibrosis. Several grade 4 nudules have coalesced to form this large mass of dense fibrosis. (Silver impregnation × 80).

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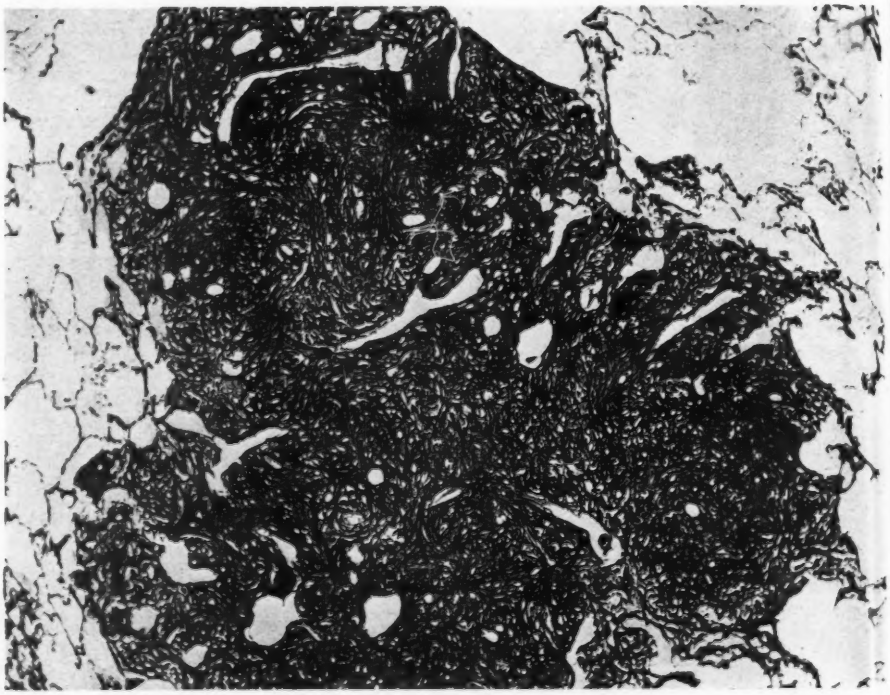

found in the lung, but fibrosis was found in the lymph nodes which were always involved where larger amounts of quartz were used. In the lungs, with 5 and 10 mg., nodules of compact reticulin were formed ; 30 mg. produced collagenous nodules ; and with 75 mg. the nodules were both collagenous and confluent.

We are grateful to the Medical Research Council for a grant to defray the expenses of this investigation. Miss V. Pash, Mr. W. Weedon and Mr. B. C. S. Hollands rendered valuable technical assistance, and Mr. E. V. Willmott prepared the photomicrographs.

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THE ACTION OF SMALL AMOUNTS OF QUARTZ AND LARGER AMOUNTS OF COAL AND GRAPHITE ON THE LUNGS OF RATS

BY

S. C. RAY, E. J. KING, and C. V. HARRISON

From the Postgraduate Medical School, London

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Experimental and field work on silicosis, while concentrating attention on the danger of exposure to the dust from hard rock, has produced a sense of false security in regard to the coal miner. Mavrogordato (1918) stated : " It may be regarded as established that all forms of dust enter the lungs and that some, such as coal, are relatively harmless, while others, such as flint, are deadly ". Bronchitis and emphysema were prevalent among old miners, but, as colliers were thought to be relatively immune to tuberculosis, it was assumed that inhalation of coal dust did not produce silicosis (Oliver, 1909 ; Collis, 1915 ; Haldane, 1931). Coal dust, as a result of experimental study, was not only claimed to be exempt from blame, but even came to be regarded as a beneficent agent capable of neutralizing the harmful effects of silica. Tattersall (1926) reported cases of silicosis among hard ground workers in coal mines, and Jousset (1928) attributed to silicosis the most important place in the production of " black lung " of coal miners. He also found by analysis that the silica content was 10 to 20 times more in the sclerosed lungs than in the normal ones. Policard and Devuns (1930), by a special microscopic technique, came to the conclusion that pulmonary anthracosis and fibrosis of the lungs of coal miners were merely two phases of the same process. Cummins (1927) advanced the hypothesis that silicosis, by causing interference with the lymph drainage of the lungs, led to the retention and accumulation of coal dust in the pulmonary tissues. Belt and King (1945) found in their experimental study that purest coal dust, whether anthracite or bituminous, had a low pathogenicity and caused only minimal fibrosis, but although results were not clear cut, there was some evidence that the pathogenicity increased with the increase of siliceous matter in the coal. Gough (1947) wrote :

> " It has now been generally acknowledged that chronic respiratory disease of South Wales coal miners is due to the inhalation of the dust encountered in everyday work in the mines or in such an occupation as loading coal in ships."

He also stated :

> " It is recognized that dust of relatively low silica content, such as commercial coal, can cause disability and fatal lung disease."

This disease was termed " coal workers' pneumoconiosis " by the Industrial Pulmonary Diseases Committee (1942) to distinguish it from classical silicosis, which may occur in those coal miners exposed to the dust of highly siliceous rocks.

In the coal industry the amount of siliceous matter inhaled varies according to the work of the miner and the part of the mine in which he is employed. Dust derived from the strata above the coal measure contains much silica ; at the coal face it consists of coal mixed with some siliceous matter, while the dust breathed by coal trimmers is composed mainly of coal.

In view of these different opinions about the pneumoconiosis of coal miners, and in order better to understand the mechanism of its production, animal experiments with mixtures of coal and silica were undertaken. King, Harrison, and Ray (1949) found that 5 and 10 mg. of quartz with 95 and 90 mg. of anthracite coal produced rather more and larger nodules, and the nodules contained more reticulin fibres than those that could be produced with 5 and 10 mg. of quartz alone. But the contrast was not always clear-cut. With most rats 5 mg. of quartz appeared to be cleared from the lungs into the lymph nodes, and likewise with several

receiving 10 mg. These animals failed to develop fibrous nodules in the lung tissue. The remainder, however, did not succeed in getting rid of their administered quartz; and the lesions which developed in their lungs were not much less severe than those in the rats which received the same amount of quartz with, in addition, 90 mg. of coal. Therefore, it was thought worthwhile to test again the tissue reactions to clean coal (anthracite with very little ash), and the same clean coal with a smaller amount (2 mg.) of quartz.

In the preceding paper it has been shown that 2 mg. of quartz is regularly cleared from rat's lungs into the lymph nodes, while with .5 and 10 mg. doses sufficient remains to produce fibrosis.

Graphite has been tested in the same way. Graphite is widely distributed in nature, and is generally found mixed with quartz (Milner, 1940). It is doubtful if pure natural graphite can be obtained commercially. Dassanayake (1948) pointed out that it is mined from vertical lodes in granite rock. According to Strong (1945) it contains 50 to 97% of carbon in natural form. Dunner and Bagnall (1946) gave the following figures for powdered commercial graphite: loss on ignition (mainly carbon) 52·7%, total silica 25·1%, and soluble silica 0·26%. A crystalline form of graphite known as "kish" is deposited in iron furnaces from molten iron on cooling.

Graphite is an important article of commerce, and in many industrial processes there is a risk of inhalation of its particles. Dunner (1945) recorded five cases of exposure to "pure graphite dust". Radiographs showed reticulation and nodulation. Dunner and Bagnall (1946) and Dunner (1948) reported a form of "snowstorm" appearance which became nodulated, and one case had a cavity. Gloyne, Marshall, and Hoyle (1949) described two cases of fatal pneumoconiosis caused by mixed dusts containing graphite, free silica, and certain silicates. In both there was progressive massive fibrosis, superimposed on dust reticulation. Harding and Oliver (1949) stated that graphite pneumoconiosis was a modified form of silicosis, and was caused by inhalation of dust composed largely of carbonaceous material with an amount of free silica of the order of 10%.

In our experiments specially prepared graphite of very low ash content was used. In experiments with graphite and quartz, the latter was the same as that previously used in the coal experiment. The size distribution, and other analyses, of the minerals used are given in Table 1.

The anthracite was powdered in an agate mortar, and was unfortunately not as fine as we should have liked. Only about 14% (by mass) was sufficiently small to be likely to be able to enter the lungs by inhalation; and if these experiments are repeated by a dusting technique it will be necessary to devise a method whereby the coal can be reduced to smaller dimensions. The graphite, on the other hand, was specially prepared in a "turbine grinder" and was 80% of respirable size, while the quartz was all sufficiently small to enter the lungs by inhalation.

TABLE 1

SIZE DISTRIBUTION AND ANALYSIS OF MINERALS

Size Group (μ)	Percentage by Number (cumulative)	Percentage by Mass (cumulative)
Anthracite A_3 Clean Coal		
Under 0·5	29·5	0·03
„ 1·0	56·6	0·26
„ 2·0	71·5	0·83
„ 3·0	82·5	2·0
„ 4·0	88·5	3·4
„ 5·0	92·5	5·1
„ 6·25	95·0	6·9
„ 7·5	97·0	10·2
„ 10·0	98·0	13·9
„ 12·5	99·5	34·6
„ 25·0	100·0	100·0
Mean particle size: by number 0·88, by mass 19·5 μ. Ash: 0·9%. Silica: 0·2%.		
Graphite		
Under 0·5	19·0	0·103
„ 1·0	38·0	0·93
„ 2·0	68·8	7·12
„ 3·0	82·0	14·45
„ 4·0	89·0	22·66
„ 5·0	94·3	34·72
„ 6·25	97·0	45·04
„ 7·5	98·5	57·99
„ 10·0	99·8	81·97
„ 12·5	100·0	100·0
„ 25·0	—	—
Mean particle size: by number 1·3, by mass 6·9 μ. Ash: 0·72%. Silica: 0·24%.		
Commercial Quartz		
Under 0·25	7·7	0·04
„ 0·5	28·9	0·8
„ 1·0	67·1	12·0
„ 1·5	83·6	20·0
„ 2·0	91·9	31·0
„ 3·0	98·0	54·0
„ 4·0	99·0	62·0
„ 5·0	99·5	70·0
„ 6·8	100·0	100·0
Mean particle size: by number 0·8, by mass 2·8 μ. Silica: 97·1%; solubility 3·4 mg./100 ml.		

Method

The animal experiments were conducted along the lines described in our preceding paper. The technique was that of Kettle and Hilton (1932) as modified by Belt and King (1945). Black and white rats of 200 g.

weight were lightly anaesthetized with ether and the trachea exposed by dissection. Suspensions in normal saline of pure coal, coal and quartz, graphite, and graphite and quartz were injected intratracheally into the lungs of rats. Each rat was given 1·5 ml. of the suspension, which contained 100 mg. of coal, 98 mg. coal + 2 mg. quartz, 100 mg. graphite, or 98 mg. of graphite + 2 mg. of quartz. For the coal experiment 32 rats were used, 16 in each group, and 40 rats for the graphite experiment, 20 in each group. The rats died or were killed at regular intervals up to 327 days. The lungs of a few rats were lost through cannibalism.

Pathological Technique.—The lungs were removed after insufflation with formol saline through the trachea. Fixation, embedding, and sectioning were carried out as in the experiments already reported. Routine sections across the middle of both lungs including the hilar lymph glands were cut in all cases, and stained with haematoxylin and eosin and Gordon and Sweets' (1936) silver impregnation method for reticulin.

TABLE 2

ASSESSMENT OF FIBROSIS IN SECTIONS OF LUNGS

Clean Coal (100 mg.)		Clean Coal (98 mg.) + Quartz (2 mg.)	
Days of Survival	Grade of Fibrosis	Days of Survival	Grade of Fibrosis
9	0	23	0
—	—	27	0
—	—	31	0
—	—	63	1
—	—	92	1
116	0	101	1
126	0	—	—
147	0	—	—
157	0	153	2
169	0	—	—
186	1 (minimal)	183	2
189	1 (minimal)	197	2
200	1	217	2
—	—	247	3
—	—	257	3
—	—	285	3
305	1	292	3
Pure Graphite (Turbine Ground) (98 mg.)		Pure Graphite (Turbine Ground) (98 mg.) + Quartz (2 mg.)	
11	0	21	0
39	0	22	0
44	0	22	0
—	—	52	1
63	0	53	1
78	1	66	1
100	1	92	1
109	1	—	—
111	1	112	1
148	1	—	—
171	1	171	2
188	1	—	—
191	1	—	—
226	1	226	2
244	1	257	2
285	1	—	—
316	1	—	—
327	1	327	2

Pathological Findings

Clean Coal (100 mg.).—On the ninth day coal was seen in coarse aggregates in groups of alveoli, and some was found in the root glands. A similar picture was seen on the 116th day. On the 126th day coal aggregates were again found, and there was some in the interstitial tissue, but no fibrosis. Lungs removed from rats on the 147th day and 157th day showed a similar picture. On the 169th day coal particles were seen in the interstitial tissue, and were just beginning to be walled-off. On the 186th and 189th days the walling-off effect of coal became distinct, but the reticulinosis still remained minimal.

Coal particles were seen enmeshed in very fine reticulin fibres at 200 and 305 days. This type of fibrosis is minimal, and we have called it Grade 1 (Fig. 1).

It was noticeable that the amount of coal in the lung parenchyma of rats killed later in the experiment was much less than in those killed earlier. This is likely to be due to elimination into the glands.

Coal (98 mg.) + Quartz (2 mg.).—In contrast to the pure coal experiment, peribronchial and perivascular reactions were noticeable as early as the 27th day. Coal particles were found enmeshed in reticulin fibrils of Grade 1 severity on the 63rd day. This picture persisted till the 101st day. From the 101st to 217th days the degree of fibrosis changed in character to Grade 2. In lungs examined from the 247th to the 292nd day the reticulin fibres became more compact and collagenous, and assumed the character of Grade 3 fibrosis (Fig. 2).

Distribution of Dust.—There was no recognizable difference in the localization of dust between the two experiments. In the early stages the dust was indifferently scattered through the alveoli. Later on, it became localized in both experiments in the vicinity of the alveolar ducts.

Graphite (100 mg.).—The histological pattern resembled that described with pure coal. The distribution was scattered, and organization was of the foreign-body type. Sections from lungs on the 11th, 39th, and 44th days showed practically no reaction. Fine reticulin fibrils enmeshing the graphite particles were first seen in the section of the lung on the 78th day. The fibrosis was of Grade 1 type. Sections from lungs from the 100th to the 327th day, at various intervals, showed that the grade of fibrosis remained the same throughout (Fig. 3).

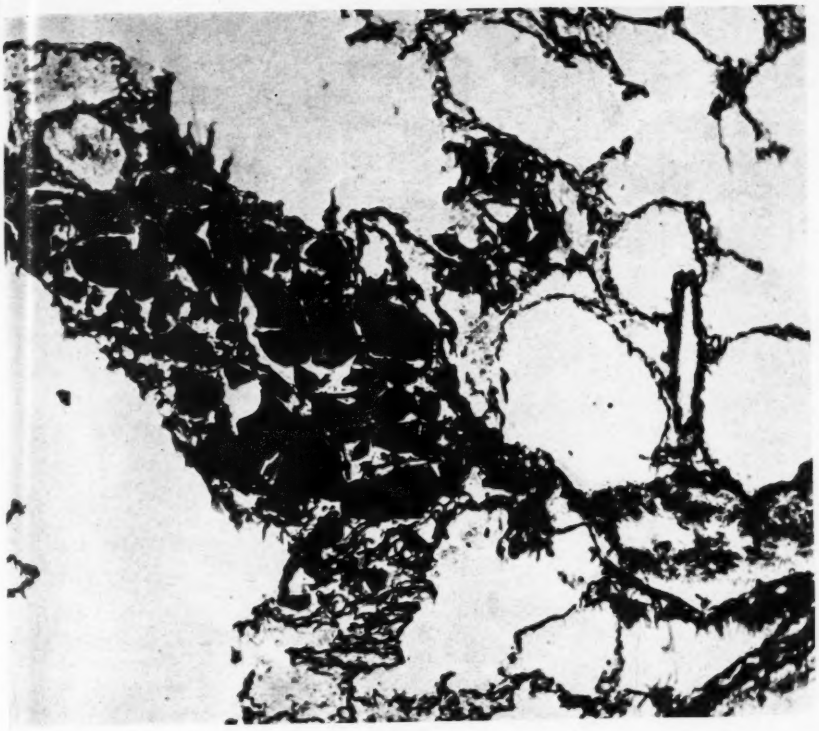

FIG. 1.—Rat lung 305 days after a dose of 100 mg. of pure coal. The coal particles are collected into a small focus and enmeshed by a minimal quantity of fine reticulin fibrils (silver impregnation ×190).

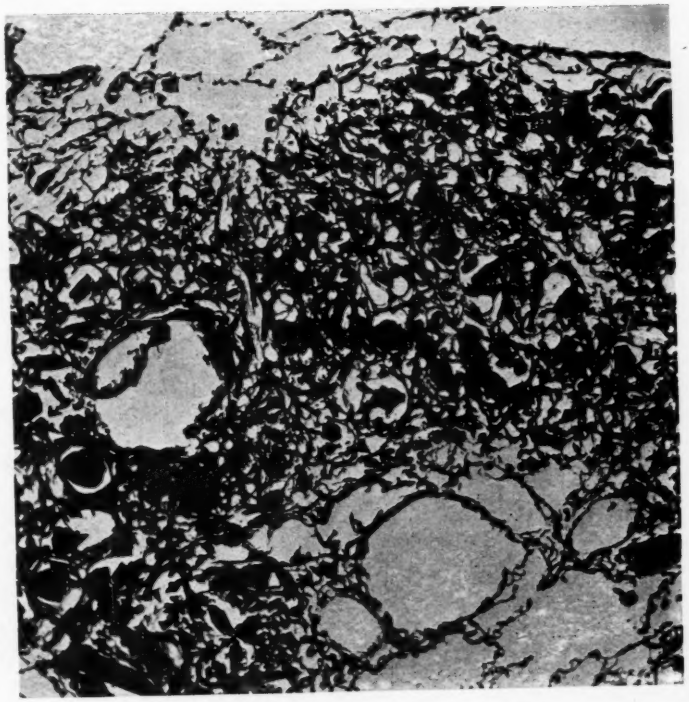

FIG. 2.—Rat lung 292 days after a dose of 98 mg. of coal *plus* 2 mg. of quartz. Compared with Fig. 1 there is considerably more reticulin production (silver impregnation × 190).

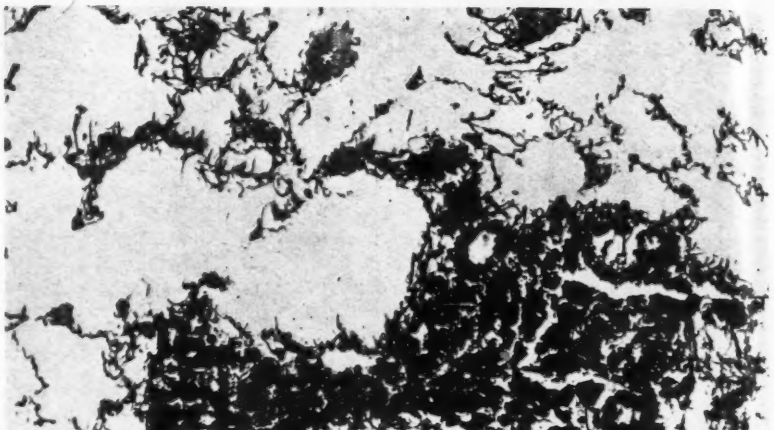

FIG. 3.—Rat lung 327 days after a dose of 100 mg. of graphite. The graphite is enmeshed in a minimal quantity of reticulin fibrils, the lesion being exactly similar to that produced by coal (Fig. 1) (silver impregnation × 190).

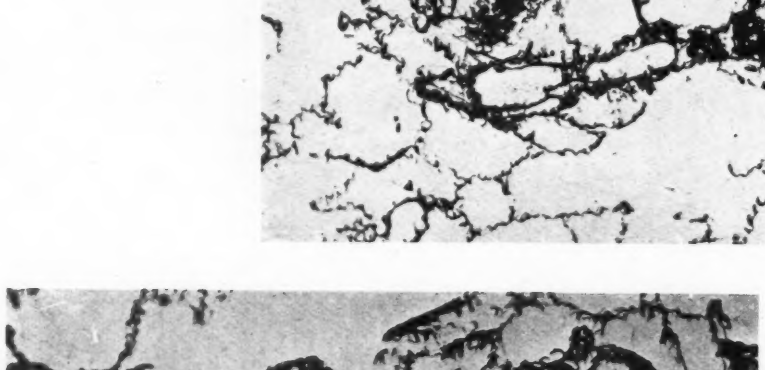

FIG. 4.—Rat lung 327 days after a dose of 98 mg. of graphite and 2 mg. of quartz. Compared with Fig. 3 there is considerably more reticulin fibril formation (silver impregnation × 190).

Graphite (98 mg.) + Quartz (2 mg.).—Grade 1 fibrosis was seen on the 52nd day. This grade of fibrosis continued till 112 days. From the 171st day onwards the degree of fibrosis changed to Grade 2 and remained so till the 327th day (Fig. 4).

Discussion

It is generally accepted that silica itself is the cause of silicosis; but the question arises as to how far non-siliceous dusts assist or retard the silicotic process, when inhaled with silica. Hollmann (1937), Böhme and Lucanus (1930) and others have concluded that pure dusts like coal and iron can be responsible for serious pneumoconiosis, as lesions produced by these dusts resemble in some respects those of silicosis. Similar observations have been made by Bergstrand (1930) and Frimann-Dahl (1937).

The effect of coal dust has been studied before, especially with reference to its power to increase phagocytosis of siliceous dusts, or to absorb the silica released in solution from such dusts. From such studies it had been concluded that inhalation of coal dust should lessen the pathogenicity of siliceous dusts (Carleton, 1924; Haldane, 1929; Whitehouse, 1938); indeed, many have believed that coal dust protects against silicosis. Sayers, Meriwether, Lanza, and Adams (1933), on the other hand, reported cases of acute silicosis from the inhalation of coal dust, and they thought the coal probably hastened the action of the silica. Policard and other French authors have noted that the lymphatics of the lung become blocked by the dust.

In this experiment we have not been able to produce any serious reaction to coal over a period of 300 days.

It has been shown in our preceding experiments with 2 mg. of silica (Ray, King, and Harrison, page 63) that this amount is almost entirely eliminated from the lung parenchyma via the lymphatics, but causes fibrosis of the hilar glands and some degree of peribronchial or perivascular fibrosis. When coal is added to this small amount of quartz, the lymphatic drainage is presumably blocked, and silica remains in the lung parenchyma. As a result the retained silica exerts its toxic effect there with the ultimate production of fibrous nodules. This appears to be the most reasonable interpretation of our results.

Pure graphite has been found to be as inert as pure coal, and exhibits no more pathogenicity in the lung than pure coal. From both these experiments it has been concluded that it is the retained silica which is pathogenic and causes the fibrous lesions in the lungs, and that the simultaneous presence of large amounts of coal or graphite exaggerates the pathogenic effect of small amounts of silica.

Summary

The part played by clean coal and pure graphite in the production of experimental silicosis has been studied.

Pure coal gives rise to no serious reaction in the animal lungs. Much of it is eliminated, and the amount retained causes only slight reticulinosis.

Small doses of quartz which alone produce no lesion in the rats' lungs, produce nodular foci of reticulinosis when large amounts of coal are added. The large amount of coal probably upsets the drainage mechanism, and the retained silica in the lung exerts its toxic effect with the production of fibrous nodules.

Pure graphite was as inert as pure coal; but fibrous nodules were produced when it was mixed with small amounts of silica. It is therefore concluded that the simultaneous presence of large amounts of coal or graphite exaggerated the pathogenic effects of small amounts of silica.

We are grateful to the Medical Research Council for a grant to defray the expenses of this investigation. Miss V. Pash, Mr. W. Weedon and Mr. B. C. S. Hollands rendered valuable technical assistance, and Mr. E. V. Willmott prepared the photomicrographs. Dr. B. M. Wright kindly obtained for us the specimen of pure graphite.

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THE ACTION OF ANTHRACITE AND BITUMINOUS COAL DUSTS MIXED WITH QUARTZ ON THE LUNGS OF RATS

BY

S. C. RAY, E. J. KING, and C V. HARRISON

From the Postgraduate Medical School, London

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Belt and King (1945) studied the tissue reactions produced in animals by selected dusts from South Wales coal mines, and found that clean coal (low ash coal fraction containing 99% coal) produced only minimal fibrosis (grade 1) with simple foreign body reaction in 250 to 500 days. In experiments with high ash coal fractions which contained from 37 to 66% coal and about 1 to 3% quartz, the rest being kaolin and mica, grade 1 fibrosis was also produced in 250 days; and the reticulin was slightly denser in character, although it could not be classified as grade 2 fibrosis. In their experiments with airborne dusts, they examined 14 samples from six different mines, which included anthracite mines with both a high and low incidence of silicosis, and also steam coal and bituminous coal mines. Nine of these samples produced only a simple foreign body reaction with grade 1 fibrosis, the average coal content of these nine samples being 85%, and the average siliceous mineral content 13%. The remaining five samples produced something more than a foreign body reaction, and fibrosis was of grades 2 and 3. These dusts averaged 74% of coal and contained 39, 28, 14, 17, and 15% (average 22%) of siliceous minerals, there being a considerable overlap between the two groups.

Further studies were made using artificial mixtures of airborne mine dusts with amounts of pure quartz which were varied from 19 to 28%, and in one case made as high as 51%. In all these experiments, at the end of one year, the fibrosis was of grade 4 type, and no difference could be found between anthracite and bituminous coal mine dusts.

In our present experiments airborne dusts from an anthracite coal mine and a bituminous coal mine have been used. These have much higher ash contents (mainly kaolin and mica) than the coal dusts used by Belt and King. They were mixed with 10% and 30% of commercial quartz of known pathogenicity in order to determine if variable amounts of quartz give variable degrees of fibrosis in the presence of these large amounts of airborne mine dusts; whether airborne dust has any modifying effect on the quartz; and, if so, whether there is a difference between the anthracite and bituminous coal in this respect.

Minerals and Materials

The ash content of the anthracite mine dust (400°C.) was 19·2%, and that of the bituminous mine dust was 25·4%. The latter was diluted with sufficient clean bituminous coal of very low ash content (from the same mine) to make the ash about 20%. The particle sizes of the two dusts were determined (Table 1). Although these were airborne dusts only about 33% (by mass) of the anthracite and 20% of the bituminous were suffi-

TABLE 1

SIZE DISTRIBUTION OF COAL MINE DUSTS

Size (μ)	Percentage of Anthracite Particles		Percentage of Bituminous Particles	
	By Number	By Mass	By Number	By Mass
Under 0·5	20·5	0·002	30·0	0·004
„ 1·0	42·5	0·1	64·5	0·14
„ 2·0	60·5	0·5	78·0	0·6
„ 3·0	73·5	1·9	86·0	1·7
„ 4·0	80·5	4·1	89·0	2·9
„ 5·0	86·0	7·6	93·0	6·2
„ 6·25	91·5	14·6	95·0	9·5
„ 7·5	94·5	21·4	96·5	13·9
„ 10·0	97·0	33·3	97·5	20·1
„ 12·5	99·0	53·4	99·0	39·6
„ 25·0	100·0	100·0	100·0	100·0
Mean particle size = 1·4 μ		Mean particle size = 0·75 μ		

ciently small to be likely to enter the lungs of miners by inhalation. The upper limit of size of respirable dust for the rat is not known ; in these experiments the dusts were introduced directly into the lung by injection.

Experimental Procedure

The animal experiments were conducted along the lines described by Kettle and Hilton (1932) and as modified by Belt and King (1945) and Ray, King, and Harrison (1951a, b). Suspensions in normal saline of mine dusts and quartz were made. These were sterilized by heating on a water bath at 100°C. for 15 minutes. The rats were lightly anaesthetized with ether, and each rat was given 1·5 ml. of the suspension intratracheally by means of a hypodermic needle. Each 1·5 ml. of suspension contained 70 mg. of mine dust (anthracite or bituminous) *plus* 30 mg. of quartz, or 90 mg. mine dust *plus* 10 mg. of quartz. Eighteen rats were used in each experiment. The rats were killed (or died) at different intervals up to 380 days, and the progress of the lesions was studied. The lungs of some rats were lost through cannibalism.

Pathological Technique.—The lungs were removed after insufflation of the trachea with formol saline. Blocks for section were taken through the middle of both lungs, and sections were stained with haematoxylin and eosin and by Gordon and Sweets' (1936) silver impregnation method for reticulin.

TABLE 2

ASSESSMENT OF FIBROSIS IN LUNGS OF RATS

Anthracite Airborne Dust (90 mg. *plus* quartz 10 mg.)		Anthracite Airborne Dust (70 mg. *plus* quartz 30 mg.)	
Days of Survival	Reticulin Fibrosis (Grade)	Days of Survival	Reticulin Fibrosis (Grade)
33	0	8	0
68	1	39	0
87	1	—	—
108	1	111	2
124	1	112	2
129	1	—	—
139	1	—	—
170	2	163	3
171	2	169	3
200	2	177	4
205	2	219	4
222	3	250	4
245	3	301	4
264	3	301	4
296	3	329	4
380	3	380	4
Bituminous Airborne Dust (90 mg. *plus* quartz 10 mg.)		Bituminous Airborne Dust (70 mg. *plus* quartz 30 mg.)	
—	—	1	0
11	0	11	0
17	0	25	0
84	1	80	2
84	1	112	4
152	2	147	4
254	3	250	4
296	4 (?)	330	4
379	3	344	4

Results

Anthracite Airborne Dust (90 mg.) plus Quartz (10 mg.).—No fibrosis was noticed on the 33rd day. Grade 1 was seen from the 68th day till the 139th day. Grade 2 fibrosis was present from the 170th day till the 205th day, and grade 3 from the 222nd day till the 380th day.

Anthracite Airborne Dust (70 mg.) plus Quartz (30 mg.).—Grade 2 fibrosis was seen on the 111th day. This changed to grade 3 on the 163rd day, and to grade 4 on the 177th day, and remained so till 380 days.

Bituminous Airborne Dust (90 mg.) plus Quartz (10 mg.).—No reticulin fibrosis was noticed till the 84th day, when the dust was found enmeshed in nodules of loose reticulin fibrils of grade 1. On the 152nd day the fibrosis had changed to grade 2. Grade 3 fibrosis was found on the 254th day and on the 379th day. One section at the 296th day showed one or two nodules bordering on grade 4.

Bituminous Airborne Dust (70 mg.) plus Quartz (30 mg.).—On the 80th day grade 2 fibrosis was noticed. Grade 4 fibrosis was present on the 112th day. Lungs examined at intervals from this day to the 344th day showed grade 4 fibrosis.

Discussion

These experiments with artificial mixtures provide a method of studying the modifying action of airborne mine dusts on different amounts of quartz of known pathogenicity. Belt and King (1945) produced grade 4 fibrosis in 365 days with 100 mg. doses of airborne mine dusts from different sources mixed with 20% or more of quartz. In the present experiment we have found that when 10 mg. of quartz was mixed with 90 mg. of either anthracite or bituminous coal the maximum effect obtained was grade 3 fibrosis after 380 days. When the amount of quartz was increased to 30 mg. mixed with 70 mg. of coal dust grade 4 fibrosis was produced as early as 112 days with bituminous, and 177 with anthracite dust, and remained so up to 380 days. The more free silica in the dust, the more severe was the pathogenicity of the dust, and no noticeable inhibitory effect of the mine dusts was demonstrated. It is interesting to compare these results with those obtained with 10 or 30 mg. of quartz alone (Ray, King, and Harrison, page 63). With 10 mg. of quartz the fibrosis produced was

grade 2 up to the 316th day, and with 30 mg. grade 3 fibrosis up to 283 days and grade 4 in 350 days. It therefore seems that these mine dusts had very little, if any, modifying or inhibitory action on quartz; rather they seem to have enhanced the action of the quartz, probably by preventing its elimination from the lung parenchyma. This is borne out by our experiments with 2 mg. of silica alone and with 98 mg. of pure coal *plus* 2 mg. of silica (Ray, King, and Harrison, page 70), where 2 mg. of silica alone was completely eliminated from the lung and produced fibrosis only in the hilar gland, whereas when the same amount was mixed with coal it produced grade 3 fibrosis in the lung parenchyma.

Summary

Tissue reactions in the lungs of rats to artificial mixtures of quartz (10 and 30 mg.) with airborne dusts (90 and 70 mg.) from an anthracite and a bituminous coal mine have been studied.

The airborne dusts contained about 20% of ash, mainly kaolin and mica. They might have been expected to modify the action of the quartz, and perhaps to different degrees for the anthracite and bituminous.

No difference was observed between the quartz-anthracite mixture and the quartz-bituminous mixture at either level of added quartz. No inhibitory effect on the action of the quartz by the mine dusts was found: rather more, than less, fibrous reaction occurred than might have been expected from the amounts of quartz used.

We are grateful to the Medical Research Council for a grant to defray the expenses of this investigation. Miss V. Pash, Mr. W. Weedon, and Mr. B. C. S. Hollands rendered valuable technical assistance. Mr. H. H. Watson kindly obtained for us the specimens of airborne mine dust.

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CARDIOVASCULAR DISEASE IN COTTON WORKERS: PART I

BY

RICHARD SCHILLING and NANCY GOODMAN

From the Nuffield Department of Occupational Health, Manchester University

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Since 1891 the Registrar General for England and Wales has recorded high death rates from cardiovascular diseases among cotton workers. Although the health of the cotton worker has been the subject of numerous investigations, there is no other evidence of either excessive mortality or morbidity from cardiovascular disease in the cotton trades.

In this paper we attempt to elucidate this problem by a further analysis of the Registrar General's evidence. In another paper the results of a detailed clinical examination of more than 300 men employed in the cotton textile mills of Lancashire will be described.

A brief description of the processes used in preparing and spinning raw cotton must be given, so that the terms used to define occupational groups can be understood. Fine cotton dust has been suggested as the cause of this high cardiovascular mortality and the degree of exposure to dust of the different groups of workers will therefore also be discussed.

Preparation and Spinning of Raw Cotton

Raw cotton arrives in this country in tightly compressed bales, which are opened in the " cotton chamber " or " opening room ". The cotton is then cleaned by removing short fibres, dust, and dirt by a pressure blowing process in the blowing room. The man in charge of this process is called the " blow room major ", and those under his charge " blow room workers ". They are exposed to fine dust and dirt emitted in the cleaning process. The cotton, now partially cleaned, passes in the form of a lap to the card room, where the cotton fibres are further cleaned and at the same time drawn out into parallel lines. This operation is done by a carding engine. The carder and his deputy (the under-carder) are in charge of the card room, in which the most important operative is the stripper and grinder. He strips the dust and cotton fibre adhering to the wire teeth of the carding engine, and sharpens the wire teeth by means of revolving rollers covered with emery.

Carding engines, which are not usually enclosed, are continually emitting dust, but during the stripping process much more dust is given off (Figs. A and B). The stripper and grinder is, of all the cotton workers, the one most heavily exposed to fine dust. The carder and under-carder are nearly always promoted strippers and grinders. After carding the cotton is prepared for spinning, which in itself is not a dusty process. But spinning rooms of the raw cotton industry are sometimes continuous with card rooms, so spinners may be exposed to dust—but not to the same extent as card and blow room workers. Weavers handle only spun cotton and have little contact with fine cotton dust and dirt emanating from raw cotton.

Respiratory Disease Among Cotton Workers

Byssinosis, a respiratory disease suffered by cotton workers exposed to dust, will be briefly described because it has influenced cardiovascular mortality.

Greenhow (1861) was one of the first British observers to describe a respiratory disease in the cotton trade characterized by difficulty in breathing. He recorded that " card room workers were very apt to become asthmatical about middle life and that few were long lived ". This disease was later named " card room asthma ", or " strippers' and grinders' asthma ". It is now called " byssinosis ". It has three stages, which were defined in the Report of the Departmental Committee on Dust in Card-rooms in the Cotton Industry (1932).

The first stage, that of irritation, usually occurs after the worker has been exposed to cotton dust for five to 10 years. He notices tightness of his chest when he returns to work after a short absence. Characteristically it affects him after the week-end

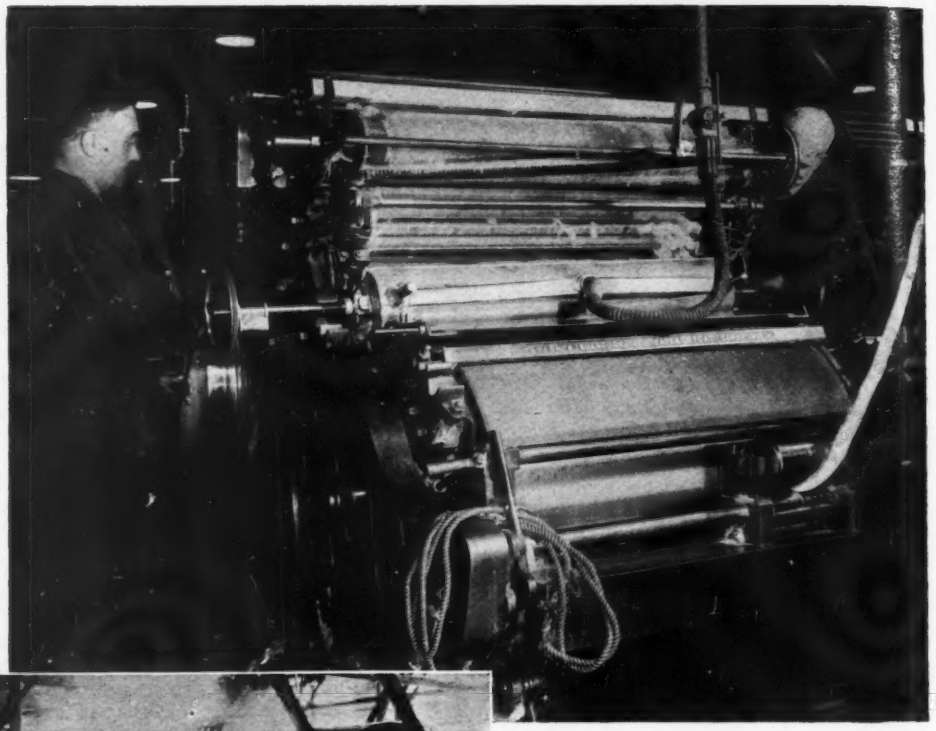


FIG. A

FIG. B

FIG. A.—Photograph of strippers and grinders brush-stripping a carding engine by the modern method with exhaust ventilation locally applied to the brush. [*Photograph by Cotton Board.*]

FIG. B.—Strippers and grinders brush-stripping a carding engine by the old methods, which had no exhaust ventilation of the stripping brush. [*From Legge's "Industrial Maladies", Oxford University Press.*]

break and hence it has been called the " Monday feeling ". The worker complains of dyspnoea, fatigue, and sometimes cough. By the next day he has recovered completely. This condition may persist without progression until he leaves the industry. Freed from exposure to dust, all symptoms cease and there is no residual disability.

In the second stage of byssinosis there is a gradual progression of symptoms, which appear on days other than Mondays, but symptoms cease when there is no exposure to dust. Many of those who reach the second stage become progressively worse and pass into the third stage—that of disabling byssinosis. Tightness of the chest and effort intolerance are now present every day and are so severe that the worker eventually has to give up his job. If he leaves it he usually has some relief, or at least the progress of the disease is checked, but effort intolerance remains a permanent disability. The aetiology of this disease is not fully understood, but the hypothesis which is usually advanced is that there is, in cotton dust, some protein allergen, either of vegetable origin, or derived from micro-organisms or fungi which are known to grow freely in the cotton and to be widely distributed in the atmosphere of card and blow rooms. The allergen, after a time, sensitizes the bronchial mucous membrane, and further exposure to dust elicits bronchospasm and possibly swelling of the bronchial mucous membrane. This condition is reversible, but in workers with a high degree of sensitivity, and after prolonged exposure, the cough and spasm give rise to emphysema, which is probably the main cause of the permanent disability.

Cardiovascular Mortality

The first reference made to cardiovascular mortality among cotton workers by the Registrar General is in his Annual Report for 1891. He found that, compared with occupied males, the mortality of cotton operatives showed an excess of one-fifth from circulatory diseases. Ten years later, in 1901, the Registrar General records a slight excess in the death rates of cotton workers from circulatory diseases and Bright's disease. Collis (1909) who was enquiring into the incidence of respiratory disease among card room workers in a clinical examination found that 91% of men working in the coarse grades of cotton were affected, but made no reference to diseases of the heart or allied conditions among these cotton workers. In his report for the triennium 1910–12, the Registrar General for the first time gives the deaths separately for two occupational groups within the cotton industry, strippers and grinders and blow room hands, instead of putting all cotton workers together. This report, being delayed by the first world war, was published in brief, and the figures are given without any comment. A further analysis of these figures, shown in Fig. 1 reveals that for all cotton workers taken together there is significant mortality excess from cardiovascular disease and nephritis over all males at 45 years of age and above, but there is no significant difference between the rates for the three occupational groups inside the cotton industry.

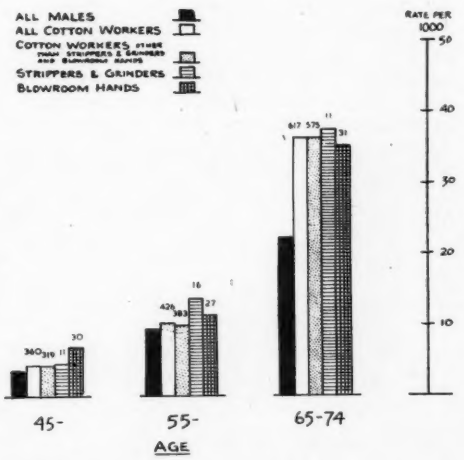
In the 1921 Decennial Supplement which refers to the triennium 1921–1923, the Registrar General reports once again high death rates for cardiovascular disease and nephritis among cotton operatives. He makes this comment. " It appears that conditions of work in textile mills promote degenerative changes of the kidneys, heart and blood vessels ". In his evidence to the Departmental Committee on Dust in Cardrooms (1932) Dr. Stevenson, representing the Registrar General, further emphasized the importance of circulatory disease as a cause of death among cotton operatives. Basing his evidence on the Registrar General's

FIGURE 1

DEATHS FROM CARDIOVASCULAR DISEASES & NEPHRITIS *

1910-1912

RATES PER 1000



* Includes acute nephritis, Bright's disease, cerebral haemorrhage apoplexy, V.D.H., angina pectoris, arteriosclerosis and other circulatory diseases.

Figures above columns are number of deaths upon which the rates are based.

reports for 1910–12 and 1921–23, he concluded that the circulatory type of disease rather than respiratory disease was the more important occupational risk for card and blow room operatives. No medical witnesses who came before this committee could express an opinion as to the inference which might be drawn from Dr. Stevenson's observations, and none apparently could support them with clinical evidence.

In the next Decennial Supplement for the triennium 1930–32, the latest published, the Registrar General reports the same trends in mortality rates for cardiovascular disease and nephritis, but he reminds his readers that these rates are high for all males resident in regions North III (York County Borough and West Riding of Yorkshire) and North IV (Lancashire and Cheshire) which contain 82% of all male textile workers (including woollen workers), but that the excess could not be explained by the presence of textile workers alone.

There have been two explanations given for the high death rates of cotton workers from cardiovascular diseases: both are apparently based on the occupational mortality statistics of the Registrar General. First, a leading article in the *British Medical Journal* (1947) contained a statement that "the inhalation of textile dusts is a steady source of death from nephritis and a group of conditions whose common factor is hyperpiesis". Second, Platt (1947a), examining the evidence on which this statement was based, suggests that the deaths from so-called nephritis are in all probability due to hypertension, and further suggests that a higher incidence of hyperpiesis could be as easily explained by hereditary factors operating in an inbred population in Lancashire cotton towns as they could by environmental influences. This suggestion is made on his own observations (Platt, 1947b), and those of others, that heredity is the most important factor in the aetiology of hypertension. In support of his thesis he points out that the increased incidence of cardiovascular disease which would be expected in the surrounding population (North IV) from this mechanism is in fact recorded by the Registrar General.

The proposition that these deaths were predominantly due to hyperpiesis is plausible. In the early 1930s the general practitioner did not often take blood pressure readings, hence hypertension might have been missed, whether as a cause of death or sickness. Moreover, it may well be the commonest single cause of heart disease over the age of 45.

In addition, this hypothesis is supported by examining the death rates of cerebral vascular disease, which is more likely than any other disease to be the end result of hyperpiesis. There is a significant excess for strippers and grinders at ages 55–64 in the 1930–32 triennium (Table 1).*

Morbidity of Cotton Workers

There have been two important enquiries into the sickness experience of cotton operatives, that of Bradford Hill (1930) and of Harvey (1939). Bradford Hill compared the sickness records of strippers and grinders and other card room workers with those of warehousemen and ring-room men in the period 1923–27. After the age of 30 the sickness rates for respiratory disease of strippers and grinders and other card room workers were two to three times as high as those of the control group of men working in the same industry but not in the dusty rooms. When respiratory diseases were excluded, the card room workers had somewhat less sickness than male workers in the ring-room and warehouse. Harvey examined morbidity rates for 1935–36 and confirmed these findings of Bradford Hill. In neither enquiry was the sickness from circulatory diseases considered separately, but there was no evidence that it was excessive.

* For 1921–23 the rates for cerebral vascular lesions cannot be given separately.

TABLE 1

DEATHS FROM CEREBRAL VASCULAR LESIONS (NUMBERS AND DEATH RATES PER 1,000) IN 1930–32

Age	All Males		Social Class III		Cotton Weavers		Cotton Spinners		Strippers and Grinders	
	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
25—	154	0·0	72	0·0	1	(0·0)†	—	—	—	—
35—	735	0·1	350	0·1	4	(0·1)	3	(0·1)	—	—
45—	3291	0·5—	1427	0·5—	13	0·5	10	0·5	—	—
55—	10621	2·0	4503	2·0	60	2·4	49*	3·4	13*	6·4
65–69	9322	5·4	3933	5·7	51	7·4	34	6·9	2	(4·9)

† Rates are in brackets when based on less than 10 deaths. * Difference statistically significant (P = ·02).

Further Analysis of the Registrar General's Mortality Figures for 1921–23 and 1930–32

Male Cotton Workers: Social Classes III and IV and All Males.—A comparison is made between the cardiovascular death rates for three principal occupations in the cotton industry for the triennia 1921–23 and 1930–32. They are compared with rates for all males and males of a corresponding social class.* Deaths from nephritis, cerebral vascular lesions, arteriosclerosis and all heart diseases have been considered together. Blow room workers could not be included as their death rates are not given separately for the triennium 1930–32 as they are for 1921–23. In the Appendix the populations, numbers of deaths and rates for each period and occupational group are given in detail (Table A). As the respective rates for the two periods show the same trends, the arithmetic means of the rates of the two triennia are given in Fig. 2 to simplify an elaborate table, but for both periods it must be stressed that the actual numbers of deaths of strippers and grinders are very small. From the age of 55 the three cotton groups have a significant mortality excess from cardiovascular disease over all males and males in Social Classes III and IV.† More interesting is the difference in the mortality rates of the three cotton groups: weavers, spinners, and strippers and grinders.

Weavers, who probably have the least exposure to dust of the three, tend to have the lowest death rates, while strippers and grinders, with certainly the greatest dust exposure, have the highest. This relation between dust exposure and death rates from cardiovascular disease makes the influence of heredity alone seem to be an unlikely cause of this high mortality. It strongly suggests an occupational influence.

In the triennium 1910–12 there is little difference between the mortality rates of strippers and grinders, blow room workers and a large group comprising the remainder of the cotton textile operatives. This appeared to be evidence against the influence of dust on mortality. When considering the development of measures for removing stripping dust, the Departmental Committee on Dust in Cardrooms reported that exhaust ventilation for stripping was introduced in some mills in 1909, but it was not until 1913 that exhaust ventilation was in general use. This may mean that dust in card rooms was excessive in the years before 1910–12 and sufficient to spread to the adjacent spinning rooms and produce its ill effects on spinners and others working in the spinning rooms near the carding engines. Thus the advantage in cardiovascular mortality of the latter group over strippers and grinders, shown in later years, may be the result of reducing the stripping dust by exhaust ventilation and preventing it reaching the spinning rooms in sufficient concentrations to produce ill effects, but not reducing it sufficiently to favour the strippers and grinders, whose cardiovascular mortality rate actually rose between 1921–23 and 1930–32. This will be discussed later.

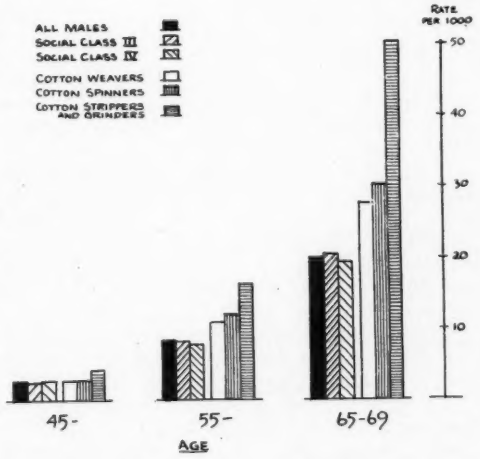
Cotton Workers and Their Wives.—A comparison of husbands' and wives' death rates is often used to measure the influence of occupation on health. For cotton workers it cannot be such a reliable index as for most other classes of worker, because the cotton operatives' wives usually work in the same industry as their husbands and are exposed to

FIGURE 2

DEATHS FROM CARDIOVASCULAR DISEASES & NEPHRITIS*

RATES PER 1000

MEAN OF 1921-3 AND 1930-2 RATES



* Includes nephritis, cerebral vascular lesions, arteriosclerosis and all heart diseases.

Note: The figures upon which this diagram is based are given in the appendix, Table A. The actual number of deaths in each triennium are small for cotton workers, particularly for strippers and grinders, and the above diagram should not be used without reference to Table A.

* All these occupations are now graded by the Registrar General as Social Class III; formerly strippers and grinders were Social Class IV. Stripping and grinding is an apprenticeable trade, and at the present time is comparable financially with the other cotton groups. In the two periods considered, however, strippers and grinders' wages were lower, and hence Social Class IV is included for comparison with them.

† This is not statistically significant for weavers aged 55–64 for 1930–32.

the same influences. But the stripper and grinder is more heavily exposed than any other worker, male or female.

In 1930–32 the rates for cardiovascular diseases for all males, cotton spinners, strippers and grinders, males in Social Class III and their wives, show that at ages 55–64 strippers and grinders appear to have a proportionally higher death rate over their wives than the other groups have over their wives. Details of these rates are given in Table B in the appendix.

Secular Changes in Death Rates.—Both respiratory and cardiovascular death rates for cotton operatives for different periods should be considered. If these deaths are directly or indirectly associated with the inhalation of dust, they should show a decline over a period of time during which dust exposure has been steadily and substantially reduced. It is well known that in the card rooms of the Lancashire cotton mills such a reduction in dust exposure has occurred from 1913 onwards. The older strippers and grinders and carders working today can remember the days when the stripper could hardly see his mate on the other side of the carding engine because of the thick clouds of dust emitted during the stripping process. They all speak of the great improvement in conditions resulting from fitting the stripping brush with an exhaust appliance, from vacuum cleaning, or from air conditioning.

Fig. 3 illustrates the secular changes in these death rates since 1910–12 for all males and strippers and grinders. Weavers' and spinners' rates can only be given for 1921–23 and 1930–32.

Respiratory deaths show a very marked decline for strippers and grinders between 1921–23 and 1930–32, while rates for all males were falling gradually. Weavers and spinners show higher rates than all males in 1921–23, but these rates had fallen to the same level as all males 10 years later. These figures seem to confirm that dust reduction has definitely had a beneficial effect on respiratory deaths of strippers and grinders. They also support the hypothesis that other cotton workers have benefited from dust reduction. Cardiovascular deaths, however, do not show the same trend. For all males, and strippers and grinders, rates rose between 1921–23 and 1930–32, but in the same period fell for weavers and spinners. Between 1910–12 and 1921–23 rates for all males were declining, while those for strippers and grinders were increasing.*

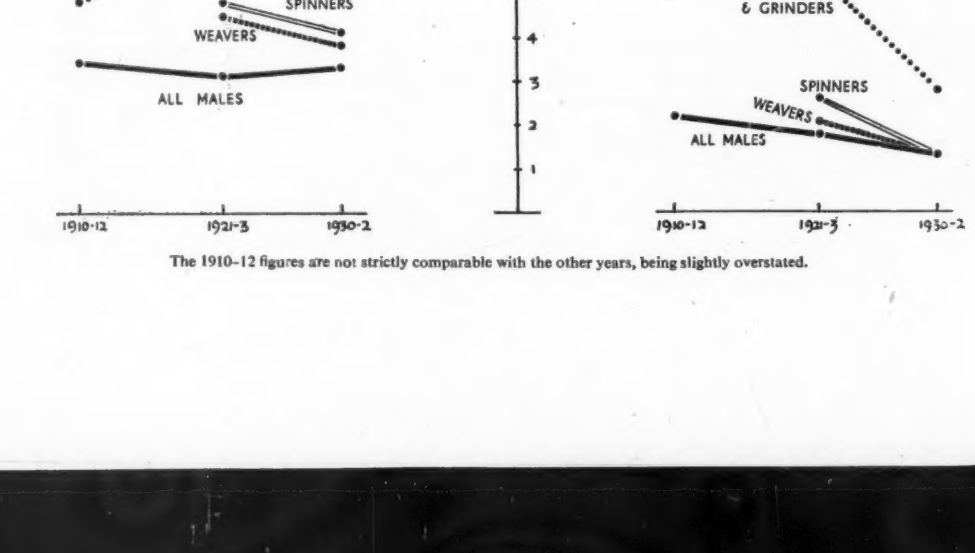
To sum up the main findings so far: the association between dust exposure and cardiovascular mortality for the triennia 1921–23 and 1930–32, and the significantly high death rates of strippers and grinders from cerebral vascular lesions, suggest that the inhalation of cotton dust may give rise to

* The rates for 1910–12 are not strictly comparable with the subsequent two periods, because "other circulatory diseases" as well as "heart diseases" are included in these years.

FIGURE 3

STANDARDISED DEATH RATES

STANDARDISED ON 1921 CENSUS ALL MALE POPULATION AGES 25–69 YEARS
RATES PER 1000



The 1910–12 figures are not strictly comparable with the other years, being slightly overstated.

ardiovascular disease and this may be hypertensive.

It would be difficult to explain this higher ortality by heredity alone because of the signifi- antly different cardiovascular mortality rates of trippers and grinders and the other cotton workers who are living and working in the same Lancashire owns.

But while respiratory death rates of strippers and grinders were declining over a period when methods of dust control were steadily improving, their cardiovascular death rates rose somewhat. This does not support the hypothesis that cotton dust causes cardiovascular disease.

The reliability of cardiovascular mortality rates and possible causes of this conflicting evidence must now be examined.

Sources of Unreliability in Mortality Rates

Many doctors certify multiple causes of death from which the Registrar General must select one for computing death rates for specific diseases. General practitioners often cannot verify their diagnoses either by necropsy or special investigation. Thus both the Registrar General and the general practitioner introduce possible sources of error into the mortality rates ascribed to specific diseases. And as the habit of multiple certification has become more common in recent years, the secular trends in the mortality rates of certain diseases may therefore be seriously misleading.

Multiple Certification.—When the medical practitioner gives more than one cause of death on the certificate the Registrar General selects one according to certain rules. In 1921–23 and 1930–32 any definite disease of the heart or kidneys was preferred to any disease of the respiratory system when both were mentioned together. In 1939 the Registrar General's selection rules were revised. Preference is now given to the disease which, as far as can be ascertained was, in the opinion of the certifying medical practitioner, the starting point of the sequence leading up to the immediate cause of death. The effect of adjusting bronchitis and cardiovascular death rates for 1921–23 and 1930–32 in the light of this revision cannot be shown accurately, but it is possible to get some idea of the likely change from two different sources—the Registrar General's statistics for 1939 (the year of change), when he classified all the death certificates according to both new and old rules, and from a study of the death certificates of card room workers, supplied by the trade unions. The latter is a source of information often available but seldom used.

The relevant conclusion of the Registrar General (1947) as regards our particular problem is :

> "Bronchitis and asthma death rates have been affected considerably . . . . by the increasing mention of myocardial degeneration as a contributory cause of death and consequent classification by the (old) rules of selection, of increasing numbers to heart disease."

He gives conversion factors so that an estimate may be made of the number of deaths before 1939 from diseases of the myocardium among the general population which would be put back to respiratory deaths if the new selection rules applied. For bronchitis in *all* males there is added 0·237 times the number of deaths which were previously ascribed to diseases of the myocardium. But for groups with a high respiratory mortality a higher proportionate transfer should be made. There is, however, another method available for the very necessary interpretation of the Registrar General's rates of cardiovascular and respiratory mortality before 1939. But it must be emphasized that it is a highly tentative procedure. In 1939, when deaths were given classified under both rules, the bronchitis deaths under the new classification were increased by transference from disease of the myocardium, endocarditis, and arteriosclerosis from amounts varying from 30% at ages 25–34 to 114% at ages 65–69. If these age-specific conversion factors are applied at the appropriate ages, the death rates from cardiovascular causes in 1930–32 are reduced by the following amounts for ages 25–69 (Tables C and E).

Weavers	8%
Spinners	10%
Strippers and grinders	24%
All males	10%

We have assumed that practitioners combined bronchitis and heart diseases on their death certificates in much the same proportions in 1930–32 as in 1939 and that the practice was similar among practitioners in cotton areas as elsewhere.* We have no means of verifying these two proportions, but if our assumptions are reasonable, cardiovascular death rates for strippers and grinders would be reduced under the new system of classification by more than twice as much as the similar rates for all males and the other cotton groups.

Death certificates of 256 card and blow room workers, or known ex-card and blow room workers, of all ages in the Rochdale, Oldham, and Bury areas, obtained from the trade unions, were examined. These covered a period from 1941 to 1948. In Fig. 4 the effect of the change in methods of classification is shown for the 25–69 age group in which there were 164 deaths.

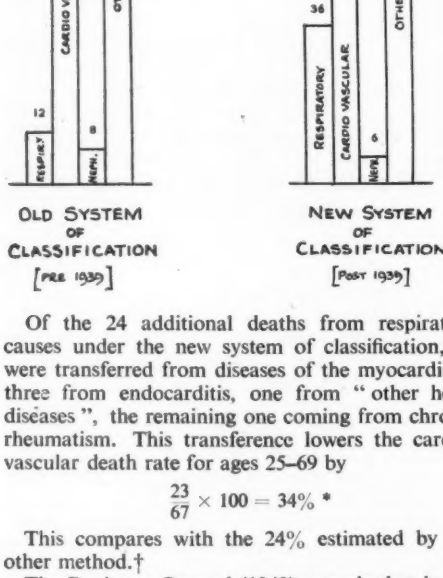
* As multiple certification continued to increase between 1930 and 1940 we may have overestimated the reduction.

FIGURE 4

CLASSIFICATION OF CAUSE OF DEATH OF 164 CARD AND BLOW ROOM WORKERS

1941 - 1948

AGES 25-69



Of the 24 additional deaths from respiratory causes under the new system of classification, 19 were transferred from diseases of the myocardium, three from endocarditis, one from "other heart diseases", the remaining one coming from chronic rheumatism. This transference lowers the cardiovascular death rate for ages 25–69 by

$$\frac{23}{67} \times 100 = 34\% \text{ *}$$

This compares with the 24% estimated by the other method.†

The Registrar General (1949) records that in the 1921–30 decennium 33% of all death certificates for non-violent deaths gave "multiple" causes. By 1935 this proportion in all death certificates had risen to 43%. It is reasonable to infer that there was a substantial increase in multiple certification between 1921–23 and 1930–32, which would mean that a higher proportion of respiratory deaths would be allocated to cardiovascular deaths in the latter than in the former triennium. This may be a possible explanation of the opposite secular trends in cardiovascular and respiratory mortality, and in fact both these death rates may have been falling.

The North IV (Lancashire and Cheshire) region in 1930–32 showed a mortality excess from respiratory diseases over all males at all ages. Some of the cardiovascular mortality excess for this region can be similarly explained. But, even after reclassification by the methods given above, death rates for heart disease and nephritis for males in North IV are still significantly greater than rates for all males at ages 45 and over, but spinners and strippers and grinders have higher rates than North IV at ages over 55 (Table D). These high cardiovascular death rates for North IV cannot be explained.‡

Errors in Diagnosis.—It is not possible to be certain about the effects on mortality rates of using vague terms to describe causes of death, or of frank errors in diagnosis. The cardiovascular group of diseases includes myocardial disease and degeneration which are vague labels used for diseases in which the exact pathology is not known. The use of "heart failure" as a cause of death is not favoured by the Registrar General, and for these diseases of uncertain pathology in which cardiac failure is the terminal result, the general practitioner undoubtedly uses "myocarditis" and the like in order to avoid enquiries about his certificates.

We have considered the reallocation to respiratory deaths of certificates on which both cardiovascular and respiratory causes are mentioned; but it is probable that there are some death certificates with no mention of respiratory disease, even though such a disease was the starting point of the pathological changes leading to death. And it is also probable that such an event occurs even more often among cotton workers exposed to dust than among all males or men of the same social class, for reasons which will now be discussed.

Byssinosis has been repeatedly described as a disease which closely simulates chronic bronchitis and emphysema, but O'Sullivan found that among 33 men suffering from disabling byssinosis 12 had no clinical evidence of bronchitis. Shaw Dunn, and Sheehan (1932) did necropsies on 10 cotton mill workers, nine of whom had been exposed to dust; while all of these nine had some evidence of

* In addition to transferring 23 deaths from cardiovascular to respiratory causes, two other deaths were transferred to the cardiovascular group from other causes, giving a net loss of 21 deaths as shown in Fig. 4.

† For all ages, the transference lowered the cardiovascular death rate by 30%.

‡ It is of interest that in 1947–8 a similar excess for heart disease is still present, in spite of the new methods of classification.

chronic bronchitis and emphysema, six showed hypertrophy of the right ventricle, four of whom died of congestive cardiac failure. Thus cotton workers with disabling byssinosis, with its characteristic asthmatic symptoms, may present a clinical picture of cardiac asthma or congestive cardiac failure, which so masks the respiratory disease that it is not mentioned on the death certificate.

For nephritis it is possible to make more definite assumptions about mistakes in diagnosis.

Platt (1947a) suggests that most of the deaths assigned by the Registrar General to nephritis in the older age groups are not renal deaths at all. In his own 161 cases for which full records were available, the main incidence of deaths from nephritis was under the age of 45. The Registrar General's figures for age incidence are totally different, with the main mortality over the age of 45. Platt's own experience is that many cases of hypertensive cardiac failure with albuminuria are diagnosed by their general practitioners as chronic interstitial nephritis.

Nephritis death rates are particularly high in certain occupations. Greenwood and Russell (1938) showed that in the 1921–23 triennium the textile trades, pottery dippers and file cutters, all of whom had high nephritis death rates also had a high risk of dying from respiratory disease.

For all occupations there is a significant correlation between the standardized death rates for bronchitis and nephritis in 1921–23 (Table 2). In 1930–32 the same type of occupations appear to have high nephritis death rates—blow room workers and foremen, strippers and grinders, and cutlers—but for this period, as shown in Table 2, there is a significant correlation between the standard mortality ratios of bronchitis and nephritis only for Social Classes III and IV. A possible explanation for this relationship is that many of the men in these trades died of congestive cardiac failure caused by respiratory disease and were certified as nephritis deaths.*

Thus there is some evidence that medical practitioners may fail to distinguish renal disease from other diseases which in their terminal stages simulate renal disease. It seems possible, therefore, that the mortality excess from nephritis in cotton workers may be due to deaths from respiratory disease, which terminates as congestive cardiac failure. If, however, occupational influences cause hypertension, as has been suggested, there will be another reason for high "nephritis" death rates in cotton workers.

Conclusions

In order to get a clearer picture of any occupational mortality excess due either to cardiovascular and respiratory diseases for the triennium 1930–32, cardiovascular and respiratory deaths are given in Fig. 5, added together, for the cotton trades, for all males, and for males of similar social classes to the cotton workers. The occupational hazard of the cotton trades, particularly of the stripper and grinder, is obvious. We know that most of this is respiratory and that in the past respiratory mortality of cotton workers has almost certainly been underestimated; but when a correction is attempted for the respiratory deaths allocated to the cardiovascular group of diseases, there still seems to be an excess of cardiovascular deaths for the cotton workers and particularly so for the strippers and grinders. Thus there may be a real excess of heart disease due to occupational influences. The latter possibility is so important in view of the very high national death rate from cardiovascular diseases and the absence of information on its relation to environmental factors that this clue must be followed up. As Morris (1951) said, cardiovascular disease of middle and old age is one of the darkest areas of vital statistics and at present the main source of information must be the mortality rates. In another paper the results of a clinical investigation of more than 300 men employed in cotton textile mills will be described.

* In the same paper Greenwood and Russell drew attention to the high nephritis death rates in barmen and inn and hotel keepers for whom an excess of alcohol is probable. In the triennia 1921–23 and 1930–32 there is a significant correlation between standardized death rates for nephritis and cirrhosis of the liver. It is possible that this correlation is due to some cases of cirrhosis of the liver which terminally have oedema being misdiagnosed as nephritis. Fishberg (1939) quoting his own experience and that of others is very sceptical of the role of alcohol in producing renal disease.

TABLE 2

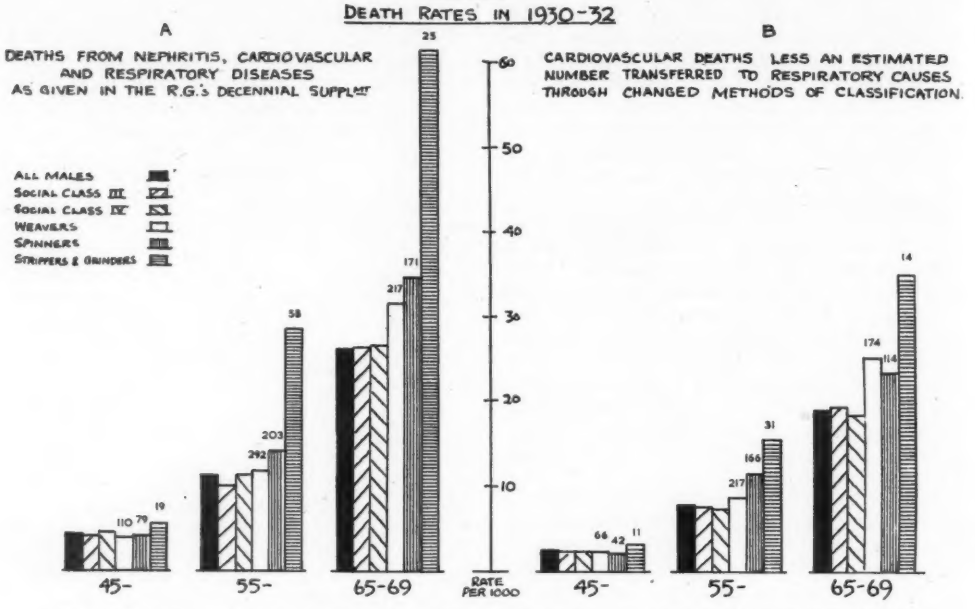
CORRELATION BETWEEN NEPHRITIS AND BRONCHITIS (AGES 20–64 YEARS)

Nephritis and Bronchitis	r	No. of Groups	P
1921–23*	$\cdot38 \pm \cdot04$	164	$P < \cdot001$
1930–32†	$\cdot22 \pm \cdot12$	71	Not significant
1930–32 (Social Class III–V)	$\cdot44 \pm \cdot13$	59	$P < \cdot01$

* From Registrar General's Decennial Supplement, based on C.M.F.'s of all trades and referring to chronic nephritis only.
† Based on S.M.R.'s of trades with 10 or more deaths, and referring to acute and chronic nephritis.

FIGURE 5

DEATH RATES IN 1930-32



Figures above columns for cotton workers are number of deaths upon which rates are based. (See Appendix, Tables C and E.)

Summary

Since 1891 the Registrar General has recorded high death rates from cardiovascular diseases among cotton workers.

It has been suggested that this mortality excess may be due to hyperpiesis caused either by the inhalation of textile dusts or by inbreeding in Lancashire cotton towns.

In this paper the Registrar General's figures are analysed principally for the triennium 1930–32 (the last to be published). It is shown that in the three occupational groups studied, strippers and grinders, weavers and spinners, there is an association between mortality and the extent of dust exposure.

The significantly high death rate of the strippers and grinders (the occupation with the highest dust exposure) from cerebral vascular lesions supports the hypothesis that hyperpiesis may be the cause of this mortality excess.

Two sources of unreliability in mortality rates are discussed. (1) Since 1920 medical practitioners have entered multiple causes of death with increasing frequency on death certificates. The Registrar General makes rules for selecting one cause of death from certificates with multiple causes. Before 1939 these rules exaggerated cardiovascular mortality and reduced respiratory mortality. Since 1939 more reliable methods of selection have been used, and some of the cardiovascular mortality of cotton workers who it is known have high respiratory death rates from byssinosis can be explained simply by methods of book-keeping. (2) The use of convenient though vague terms such as " myocarditis " to describe causes of death, and errors in diagnosis, particularly of nephritis in the older age groups, may also exaggerate cardiovascular death rates.

When all possible corrections are made for the exaggeration of cardiovascular mortality through the erroneous allocation of respiratory deaths to it, there still remains an excess of cardiovascular mortality particularly for strippers and grinders. This may well be real and may be due to occupational influences.

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tion, for giving us access to trade union records of death certificates; Mr. W. Hart of the Manchester Public Health Department for assistance in classifying death certificates; the Cotton Board for taking a photograph of modern methods of brush stripping and for permission to publish it; and the Oxford University Press and Dr. S. A. Henry for permission to reproduce the photograph of old methods of brush stripping from Legge's "Industrial Maladies".

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APPENDIX

In the Appendix which follows the information discussed in the text is tabulated and arranged for comparison with the figures given by the Registrar General.

Table A. Deaths from Cardiovascular Diseases and Nephritis, 1921–23 and 1930–32.

Table B. Deaths from Cardiovascular Diseases and Nephritis for Males Compared with Wives. 1930–32.

Table C. Deaths from Cardiovascular Diseases and Nephritis. 1930–32.
- (*a*) As given by the Registrar General.
- (*b*) After correction for new system of classification.

Table D. *Deaths from Heart Diseases and Nephritis. 1930–32.
- (*a*) As given by the Registrar General.
- (*b*) After correction for new system of classification.

Table E. Deaths from Respiratory Diseases. 1930–32.
- (*a*) As given by the Registrar General.
- (*b*) After transfer of cardiovascular deaths to respiratory deaths by applying correction for new system of classification.

* Heart diseases and nephritis are given so that the necessary comparisons can be made with North IV for which the Registrar General does not give all cardiovascular deaths.

TABLE A

DEATHS FROM CARDIOVASCULAR DISEASES AND NEPHRITIS (NEPHRITIS, CEREBRAL VASCULAR LESIONS, ARTERIOSCLEROSIS, HEART DISEASE, INCLUDING V.D.H.) IN THE PERIODS 1921–23 AND 1930–32

Age Group ..	25–		35–		45–		55–		65–69	
Years	1921–23	1930–32	1921–23	1930–32	1921–23	1930–32	1921–23	1930–32	1921–23	1930–32
All Males :										
Population ..	2621280	3062282	2496375	2512356	2133179	2302873	1382843	1765509	449363	577970
Deaths (3 Years) .:	4365	3249	7731	6705	16804	19213	33816	46020	25097	37023
Annual death rate (per 1,000) ..	0·6	0·4	1·0	0·9	2·6	2·8	8·2	8·7	18·6	21·4
Social Class III										
Population ..	1176883	1575602	1075975	1220628	846809	1036087	504706	733163	153053	231536
Deaths (3 Years)..	1860	1560	3014	3041	6014	8161	12126	18751	8845	15045
Annual death rate (per 1,000) ..	0·5	0·3	0·9	0·8	2·4	2·6	8·0	8·5	19·3	21·7
Social Class IV :										
Population ..	514558	568620	465707	413230	407169	375170	269545	300436	85742	98144
Deaths (3 Years)..	852	621	1444	1176	3097	3114	6086	7602	4518	6266
Annual death rate (per 1,000) ..	0·6	0·4	1·0	0·9	2·5	2·8	7·5	8·4	17·6	21·3
Cotton Spinners :										
Population ..	9201	11720	7687	7159	6058	6353	3718	4819	793	1646
Deaths (3 Years)..	34	15	31	18	52	50	133	178	78	135
Annual death rate (per 1,000) ..	1·2	0·4	1·3	0·8	2·9	2·6	11·9	12·3	32·8	27·3
Cotton Weavers :										
Population ..	11819	10729	10294	9537	9563	9071	5385	8230	1595	2310
Deaths (3 Years)..	34	14	32	30	81	72	198	237	132	191
Annual death rate (per 1,000) ..	1·0	0·4	1·0	1·0	2·8	2·6	12·3	9·6	27·6	27·6
Strippers & Grinders :										
Population ..	1692	1426	1333	1462	907	1139	480	679	117	136
Deaths (3 Years)..	4	2	4	3	14	12	19	40	18	20
Annual death rate (per 1,000) ..	(0·8)	(0·5–)	(1·0)	(0·7)	5·1	3·5	13·2	19·6	51·3	49·0

Population = Census population of 1921 or 1931. Death rate = Annual rate per 1,000 population. Deaths = Total deaths for the 3 years 1921–23 or 1930–32.

Significance (level taken $P < .05$)

1921-23

Strippers and Grinders significantly different from all groups except other cotton workers, at ages 45–, 55–, and 65–69 ($P < .01$). Strippers and Grinders *not* significantly different from spinners at age groups 55– ($.7 > P > .5$) or 65–69 ($.1 > P > .05$) or at these two ages together ($.1 > P > .05$).

Strippers and Grinders *not* significantly different from weavers at age 55– ($.8 > P > .7$) but is significant at ages 65–69 ($.05 > P > .02$). Weavers significantly different from non-cotton groups at ages 55– and 65–69 ($P < .01$).

Spinners significantly different from non-cotton groups at ages 55– and 65–69 ($P < .01$). Spinners and weavers not significantly different at any age.

1930-32

Strippers and grinders significantly different from *all* groups at ages 55–, 65–69 ($P < .01$).

Spinners significantly different from non-cotton groups at ages 55– and 65–69 ($P < .01$). Weavers significantly different from non-cotton groups at ages 65–69 ($P < .01$), but not significantly different from non-cotton groups at ages 55–64 (e.g., all males ($.2 > P > .1$)). Spinners and weavers not significantly different at any age group except 55–64 ($P < .01$).

TABLE B

DEATHS FROM NEPHRITIS, CEREBRAL VASCULAR LESIONS, ARTERIOSCLEROSIS, HEART DISEASE (INCLUDING V.D.H.) IN THE PERIOD 1930–32 COMPARED WITH DEATHS OF WIVES

Age Group	All Males		All Married Women		Social Class III				Cotton Spinners				Strippers and Grinders			
					Males		Wives		Males		Wives		Males		Wives	
	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate
25–	3249	0·4	2751	0·4	1560	0·3	1381	0·4	15	0·4	19	0·9	2	(0·5–)	1	(0·3)
35–	6705	0·9	5595	0·8	3041	0·8	2726	0·8	18	0·8	19	1·0	3	(0·7)	3	(0·7)
45–	19213	2·8	13088	2·3	8161	2·6	5872	2·3	50	2·6	57	3·5	12	3·5	11	4·0
55–	46020	8·7	23775	6·5	18751	8·5	10247	6·5	178	12·3	95	9·2	40	19·6	15	11·3
65–69	37023	21·4	15011	15·7	15045	21·7	6434	16·0	135	27·3	76	32·4	20	49·0	7	(33·3)

The figures for weavers' wives are not available. Number of deaths = total for 1930–1932. Rate = annual rate per 1,000.

TABLE C

DEATHS FROM CARDIOVASCULAR DISEASES AND NEPHRITIS AND DEATH RATE PER 1,000 POPULATION IN THE PERIOD 1930–32

	Age	(*a*) Cardiovascular and Nephritis Deaths as in the Registrar General's Decennial Supplement		Number Transferred to Bronchitis*	(*b*) Cardiovascular and Nephritis Deaths after Transference	
		Number	Rate	Number	Number	Rate
All Males	45–	19213	2·8	1861	17352	2·5
	55–	46020	8·7	4423	41597	7·9
	65–69	37023	21·4	4082	32941	19·0
Social Class III	45–	8161	2·6	752	7409	2·4
	55–	18751	8·5	1735	17016	7·7
	65–69	15045	21·7	1628	13417	19·3
Social Class IV	45–	3114	2·8	371	2743	2·4
	55–	7602	8·4	955	6647	7·4
	65–69	6266	21·3	886	5380	18·3
Spinners	45–	50	2·6	7·8	42·2	2·2
	55–	178	12·3	11·6	166·4	11·5
	65–69	135	27·3	20·6	114·4	23·2
Weavers	45–	72	2·6	6·4	65·6	2·4
	55–	237	9·6	18·4	216·6	8·8
	65–69	191	27·6	17·1	173·9	25·1
Strippers and Grinders ..	45–	12	3·5	1·4	10·6	3·1
	55–	40	19·6	8·7	31·3	15·4
	65–69	20	49·0	5·7	14·3	35·0

* Based on assumptions mentioned on page 83 in the text.

TABLE D

NUMBER OF DEATHS AND RATES PER 1,000 FROM HEART DISEASES AND NEPHRITIS IN THE PERIOD 1930–32

Age	All Males		Social Class III		North IV, 1931–1932		Weavers		Spinners		Strippers and Grinders	
	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate
	(*a*) As given by the Registrar General											
25–	3088	0·3	1484	0·3	392	0·4	13	0·4	15	0·4	2	(0·5)
35–	5927	0·8	2665	0·7	734	0·9	26	0·9	14	0·7	3	(0·7)
45–	15592	2·3	6579	2·1	2007	2·8	58	2·1	40	2·1	11	3·2
55–	33907	6·4	13662	6·2	4088	7·5	173	7·0	123	8·5	24	11·8
65–74	54381	19·0	22044	19·3	5998	22·5	291	26·8	193	26·8	22	31·5
	(*b*) After re-classification*											
25–	2985	0·3	1442	0·3	373	0·4	12	0·4	15	0·4	2	(0·5—)
35–	5439	0·7	2461	0·7	653	0·8	24	0·8	10	0·5—	2·5	(0·6)
45–	13760	2·0	5838	1·9	1696	2·4	52	1·9	32	1·7	9·6	(2·8)
55–	29667	5·6	11999	5·5–	3462	6·3	155	6·3	112	7·7	15·6	7·7
65–74	47562	16·6	19231	16·8	4837	18·2	256	23·6	163	22·7	15·4	22·0

* Re-classification based on assumptions mentioned on page 83 in the text. North IV significantly different from all males for all age groups 45 and over ($·01>P>·001$). Spinners significantly different from North IV for age groups 55–64 ($·01>P>·001$). and 65–74 ($·05>P>·01$). Weavers significantly different from North IV for age group 65–74 ($·01>P>·001$). Strippers and grinders *not* significantly different from North IV for all separate age groups or for age groups 45–74 together ($P>·05$).

TABLE E

DEATHS FROM RESPIRATORY DISEASES (EXCLUDING RESPIRATORY TUBERCULOSIS) IN THE PERIOD 1930–32

Age	(*a*) As Given by the Registrar General				(*b*) After Transference* from Cardiovascular Diseases			
	All Respiratory Diseases		Bronchitis		All Respiratory Diseases		Bronchitis	
	No. of Deaths 1930–32	Rate per 1,000 per Year	No. of Deaths 1930–32	Rate per 1,000 per Year	No. of Deaths 1930–32	Rate per 1,000 per Year	No. of Deaths 1930–32	Rate per 1,000 per Year
All Males :								
25–	3148	0·3	339	0·0	3251	0·4	442	0·0
35–	5889	0·8	1012	0·1	6377	0·8	1500	0·2
45–	10357	1·5	2632	0·4	12218	1·8	4493	0·7
55–	13180	2·5—	4569	0·9	17603	3·3	8992	1·7
65–69	7897	4·6	3571	2·1	11979	6·9	7653	4·4
Social Class III :								
25–	1392	0·3	137	0·0	1434	0·3	179	0·0
35–	2596	0·7	424	0·1	2800	0·8	628	0·2
45–	4307	1·4	1064	0·3	5059	1·6	1816	0·6
55–	5501	2·5	1792	0·8	7236	3·3	3527	1·6
65–69	3088	4·4	1424	2·1	4716	6·8	3052	4·4
Social Class IV :								
25–	678	0·4	71	0·0	700	0·4	93	0·1
35–	1104	0·9	214	0·2	1207	1·0	317	0·3
45–	1891	1·7	525	0·5—	2262	2·0	896	0·8
55–	2538	2·8	987	1·1	3493	3·9	1942	2·2
65–69	1527	5·2	775	2·6	2413	8·2	1661	5·6
Spinners :								
25–	12	0·3	1	(0·0)†	12·3	0·4	1·3	(0·0)
35–	21	1·0	8	(0·4)	24·9	1·2	11·9	0·6
45–	29	1·5	11	0·6	36·8	1·9	18·8	1·0
55–	25	1·7	12	0·8	36·6	2·5	23·6	1·6
65–69	36	7·3	18	3·6	56·6	11·5—	38·6	7·8
Weavers :								
25–	15	0·5—	4	(0·1)	16·2	0·5	5·2	(0·2)
35–	29	1·0	3	(0·1)	30·4	1·1	4·4	(0·2)
45–	38	1·4	9	(0·3)	42·4	1·6	15·4	0·6
55–	55	2·2	19	0·8	73·4	3·0	37·4	1·5
65–69	26	3·8	15	2·2	43·1	6·2	32·1	4·6
Strippers & Grinders:								
25–	2	(0·5–)	0	(—)	2	(0·5—)	0	(—)
35–	4	(0·9)	1	(0·2)	4·5	(1·0)	1·5	(0·3)
45–	7	(2·0)	2	(0·6)	8·4	(2·5—)	3·4	(1·0)
55–	18	8·8	9	(4·4)	26·7	13·1	17·7	8·7
65–69	5	(12·3)	5	(12·3)	10·7	26·2	10·7	26·2

* In 1939, when deaths were given for both systems of classification, bronchitis deaths under the new system were increased by transference from disease of the myocardium, endocarditis, and arteriosclerosis by the following amounts : 25–, 30·5%, 35–, 48·2% ; 45–, 70·7% ; 55–, 96·8% ; 65–69, 114·3%. These are the conversion factors used here.

† The rates in brackets are based on less than 10 deaths.

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AN INFLUENZA EPIDEMIC IN INDUSTRY

BY

A. LLOYD POTTER

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The recent influenza epidemic in Britain first appeared in Tyneside in the latter half of December, 1950, and is believed to have had its origin in Scandinavia (*Lancet*, 1951). Shortly afterwards there was an outbreak in Belfast, and almost concurrently in Merseyside, where the towns of Widnes and Runcorn, the centre of the chemical industry, were particularly affected. The epidemic received much publicity in the press, and this district was wrongly assumed by many to be the starting point of the outbreak in Britain.

This paper is a brief account of the effects of the influenza epidemic in six chemical works in the Widnes and Runcorn area with a total population of 7,928. The processes in these factories are, in the main, continuous, and the factories must, at all costs, be prevented from closing down.

The doctor in industry, and particularly in a chemical works, must always be on the watch for toxic manifestations related to plants or processes. However, in a chemical works such manifestations are relatively uncommon, and much of the doctor's time is devoted to the diagnosis and preliminary treatment of constitutional illness to which anyone is subject. My attention was drawn to the symptoms which appeared on December 18 in the six factories coming under my supervision. By noon in one of the factories 14 persons had reported to the medical department complaining of headache, aching limbs and back, dry throat, tracheitis, and pyrexia up to about 102°F. All appeared to be suffering from typical influenza. A rapid check proved that the cases were not all concentrated in one part of the works, but were coming from widely separated sections. The other five factories had the same experience and there was no doubt that the disease was rapidly reaching epidemic proportions. Little could be done to treat the patients in the factory medical departments, which became clearing houses for diagnosis and transmission of patients exhibiting the typical symptoms. The factory car services were fully occupied in conveying patients to their homes and delivering notes from the works medical officers to the general practitioners.

It is often impossible to distinguish clinically between influenza A and undifferentiated acute respiratory disease, as the Commission on Acute Respiratory Diseases (1948) found in their investigation of an influenza A epidemic in military training camps in North Carolina. In the cases seen in our medical departments pyrexia had to be the chief factor in the decision to send a patient home or allow him to continue at work.

Within a few days, no doubt because their own staff were also depleted, the local press reported the story of the influenza wave and its effects on the factory population, and this was soon taken up and expanded, in no uncertain terms, by the national press. It became headline news and drew the anticipated answer from medical authorities that, in fact, no epidemic existed. In spite of this, patients continued to arrive in ever increasing numbers at the factory medical department and, in turn, general practitioners became more and more hard pressed. The publicity continued and so alarmed the population both in and out of the factories that the figures for sickness absence rose alarmingly. Ordinary colds, minor pharyngitis, headaches and lumbago, were all labelled influenza, and the overworked practitioners with queues outside their surgeries could not possibly be accurate in diagnosis or certification. Their chief preoccupation was to decide which patients required their attention and which could look after themselves. It was quite impossible for them to visit all the sufferers, and, as one doctor observed, he was only able to prescribe on the telephone and follow up with a visit if the temperature did not subside in three days.

The incidence steadily increased until the end of the first week in January, 1951, but, thereafter, the numbers fell steadily, while other parts of the country felt the impact of the epidemic and the health authorities agreed that the disease had indeed reached minor epidemic proportions.

Symptomatology

The disease, fortunately, was of a mild type, and uncomplicated cases exhibited the typical symptoms of fever, headache, aching back and limbs, pharyngitis and tracheitis. Pyrexia continued for about 72 hours and the patient was left in a debilitated and depressed condition, with bronchitis which persisted for about two to three weeks. The average duration of sickness absence in uncomplicated cases was 14–21 days, the longer period of absence being necessary for manual workers. The incidence of bronchopneumonia was high in the older age groups, and in the general population the death rate from influenzal complications rose steeply in old people.

Distribution of Cases

In the chemical industry it is uncommon to have large groups of workers employed on any one process. Such groups are only found in offices, in general services workshops, and in packing and distribution sections. This fact probably played a part in limiting the spread of infection by direct contact, and it was noticeable that in one factory the highest incidence was in a packing section staffed by 121 girls. Here the figure for absenteeism rose on New Year's day to 47·1% compared with 24·7% elsewhere in the same factory. Absenteeism from causes other than sickness is to be expected on this day, but the comparison between different departments should be valid. The corresponding figure for the office staff rose to 13·1% only, in spite of the greater crowding together and contact which, of necessity, occurs in large offices.

These figures appear to contradict the suggestion that a high incidence may be expected in more crowded places, and it is a matter for speculation why there should be such a difference between the office staff and the packing section. The latter is largely a population of young girls, while the office staff is composed of 75% males and 25% females. The girls are employed on repetitive jobs and, although working on an incentive bonus scheme, may be expected to take an unofficial holiday at New Year more readily than would the office staff. This point is confirmed by a comparison of the figures for January 3, when 28·9% were absent from the packing section and 13·7% from the offices. Moreover, it is possible that the unfortunate publicity given to the epidemic influenced the more youthful employees to absent themselves for minor complaints, for which, normally, they would obtain treatment in the works medical department.

Fig. 1 shows the daily number of absentees in the six Merseyside factories during the period December 11, 1950, to January 25, 1951. The

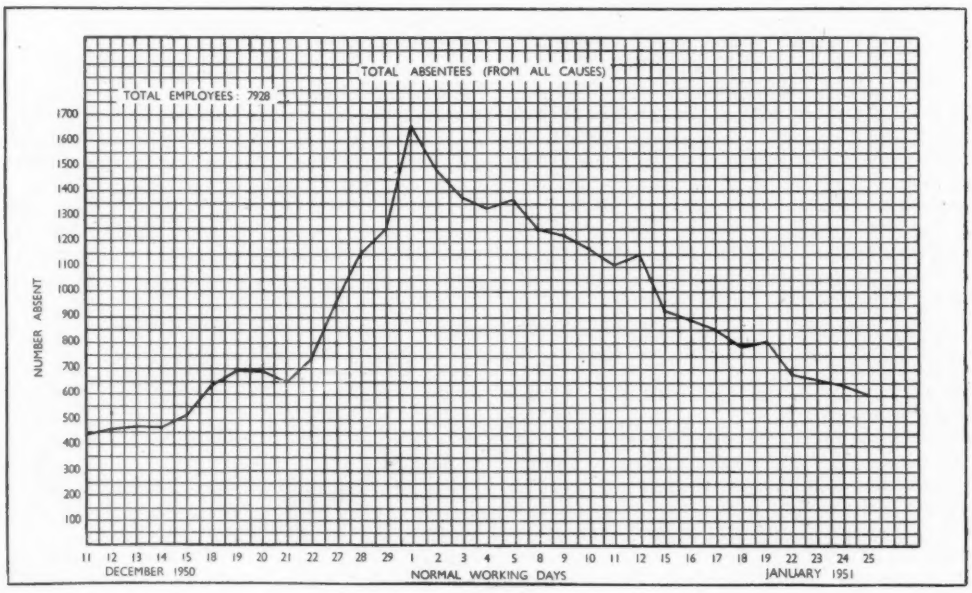

FIG. 1

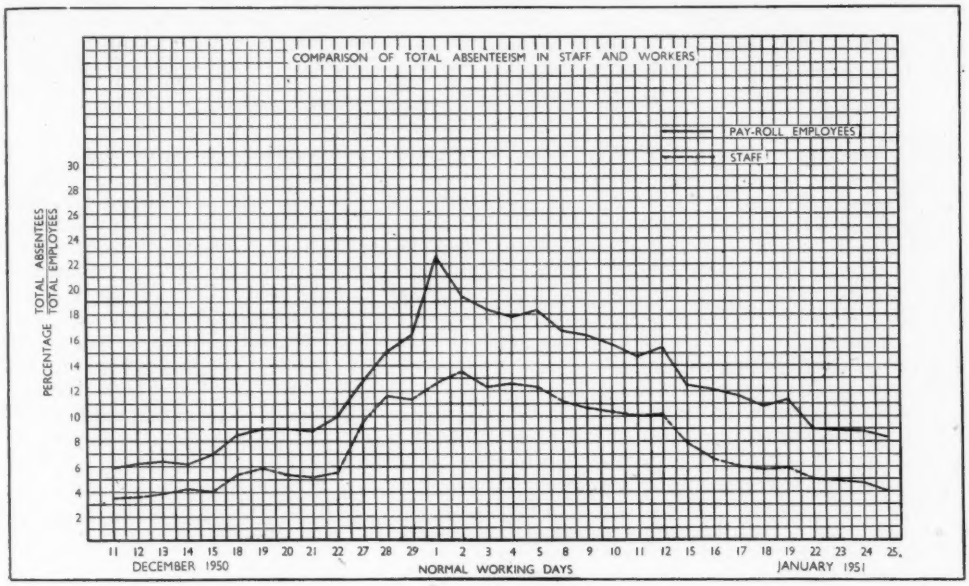

FIG. 2

effect on industry has perforce to be estimated by total absenteeism from all causes, and it must, therefore, be realized that the figures shown in the graph refer to this and not solely to absence from influenza. Normal absenteeism from all causes in the same factories averages 6% (i.e. about 500 persons), at this time of the year, and it can be assumed that the difference between this figure and those shown are almost entirely due to the epidemic or fear of it.

Fig. 2 compares the percentage of staff and pay-roll* employees absent each day. The latter normally have a higher absence rate, possibly due to their shift hours, exposure to cold and wet weather, and the greater physical effort demanded by their work. It is suggested that the proportionately higher absence of pay-roll workers during the height of the epidemic was because they were more prone than staff to treat minor complaints as influenza.

Effect on Industry

The wide distribution of cases and the natural dispersal of employees in the chemical industry fortunately prevented any necessity for closing down processes, and there was, consequently, no loss of production. The epidemic, however, gave rise to a considerable amount of overtime, and many men were required to work double shifts in order to keep processes going. Occasional double shift and overtime working have no ill effects, but when continued indefinitely there must be a lowering of resistance, due to fatigue, which is a probable factor in the further spread of this type of infection. It is interesting to note that those workers who suffered from typical colds, with only naso-pharyngeal symptoms, did not develop true influenza, and were able to continue at their jobs. Thus, early observation and reassurance of such cases was an important factor in preventing unnecessary absence. The short duration of absence in uncomplicated cases also played an important part in the maintenance of full production, for if absence had been unduly prolonged, there is little doubt that the fatigue and lowered resistance experienced by those who did not themselves suffer from the disease would eventually have led to a greater disruption of normal working.

The numbers involved in the epidemic were the highest recorded for any reason for very many years in this industry. In spite of the mild type of disease, therefore, the effects could have been very serious if the peak had not been passed so quickly and if those who remained at work had not so willingly shouldered the extra burden.

* Pay-roll employees are mostly manual workers, a minority of whom are paid full wages while they are absent.

Prophylaxis

Many requests were passed to the medical department by management and workers for some means of combating the spread of infection within the factories. Careful consideration was given to suggestions for spraying process buildings and offices with bactericidal solutions, to the issue of antiseptic tablets, to regular attendance for gargles, and to many other similar ideas. None of these measures has been proved to have any effect in preventing the spread of influenza. When it is realized that most people in industry spend less than one-third of their time at work, and the other two-thirds at home, in cinemas and places of entertainment, in buses and trains, shops, football matches and the like, it is probable that any method intended to obviate or reduce the chance of becoming infected at work will have little effect on the total incidence of the disease. It is surely true to say that influenza is spread more readily in public transport or in crowded places of entertainment than in well ventilated, airy process buildings in a chemical works. True, there is a considerable psychological effect produced by spraying buildings and telephones and by issuing some form of antiseptic lozenge. After due consideration, therefore, our views on the matter were made plain to both workers and management through the medium of the joint consultation committees, and no such measures were introduced. All employees, however, were advised to avoid crowds as much as possible, and to report to the works medical department if any of the recognized symptoms of the disease were experienced. The short incubation period of influenza can ensure the early removal of a sufferer from contact with others, and this will probably control the spread of infection much more positively than the use of the preventive measures which a community usually demands. Though such measures may be classed as excellent showmanship there is no evidence that they have any real effect in retarding the intensity of an epidemic.

The doctor in industry may at first sight appear to be quite helpless in controlling an epidemic of influenza in his factories. If, however, his efforts are concentrated on early diagnosis of the disease, preliminary treatment, and the removal of the infectious persons from the factory to the care of their own practitioners in their own homes, he will have done much to prevent the spread of the disease. Moreover, if he can then advise employees on general precautions to be taken and can separate the true cases of influenza from those who only think they have contracted the disease, he will have done much to maintain normal production in the factory.

Summary

A short description is given of the recent epidemic of influenza which attacked the Merseyside towns of Widnes and Runcorn.

The effects on workers and staff in the chemical industry are discussed.

Attention is drawn to the dangers of publicity in epidemics of this type.

The value or otherwise of prophylactic measures within the factory is discussed, and the role of the industrial medical officer in an influenza epidemic is suggested.

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FIG. 1.—At least 24 hours after stopping work the sweating is still profuse, and it coincides exactly with the hyperaemia.

FIG. 2.—The affected area is sharply limited and corresponds precisely to the part of the hand in contact with the vibrating surface in its grasp.

FIG. 3.—The photograph shows the hand in the position characteristic of the job (tip-stretcher). Sweating is still conspicuous on the hypothenar pad.

FIG. 1

FIG. 3

FIG. 2

[*Facing page* 95

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A NEW INDUSTRIAL DISEASE CALLED HYPERHIDROSIS ERYTHEMATOSA TRAUMATICA

BY

J. H. TWISTON DAVIES

From the Manchester and Salford Hospital for Skin Diseases

(RECEIVED FOR PUBLICATION JANUARY 23, 1951)

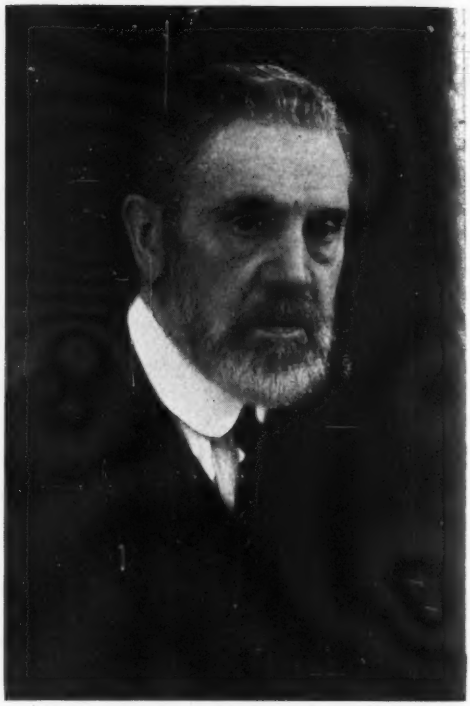
In the past and in the less heavily industrialized parts of the country managements were never quite as resigned as they are today to " dermatitis ". Nor was any and every case of skin disease accepted by the works manager as dermatitis just because it occurred in a manual worker, however uncritically it might have been accepted on purely clinical grounds by the doctor. Thus it happened early in 1940, at a time when all sorts of small firms were undertaking the manufacture of shell fuse caps, that the occurrence of a group of cases of " dermatitis " said to be caused by lubricating oil attracted sufficient interest for a dermatologist to be consulted.

Twelve capstan lathes had been set up in the first floor of a building which, though part of a garage, had not been used as a machine shop before. The job consisted of cutting screw threads in some steel part or other of a fuse cap, but because the steel contained an alloy to facilitate cutting, the only lubricant used was ordinary engine oil. What the works manager knew and the certifying factory surgeon did not was that the so-called " dermatitis " affected only those men who operated three particular lathes. Indeed one man who disregarded his doctor's advice never to touch oil again and carefully to avoid all dust and liquids, returned to work on one of the other machines, and had no recurrence, whereas another man who had been doing exactly the same job for six months without trouble took his place and at once developed the disease. The works manager therefore wanted me to find out for him what was the " juju " attached to these particular lathes. A loose joist under the flooring quickly supplied the answer, for the condition shown in Figs. 1, 2, and 3 is the effect of vibration and not of cutaneous sensitivity to an external irritant. Indeed it appears to have something in common with the familiar red patches on the soles of the feet of soldiers with hyperhidrosis who, in the pursuit of their calling, repeatedly stamp their feet on hard pavements. The main difference is that in the syndrome I am describing only the red areas sweat, and the signs disappear within a week. Since I have practised in the north I have seen two or three cases of this condition in " tip stretchers ". These are hatters whose job it is to hold down a cone of felt for it to be beaten into the shape of a hat by an artificial head which bobs up and down with a great clatter. Fig. 3 shows not only the hyperhidrosis erythematosa traumatica but also the characteristic callosities of a " tip stretcher ".

I am told on good authority that the condition has not hitherto been described, so I venture to submit this short note and to propose the sort of name for the condition that would naturally be expected of a dermatologist.

OBITUARY

H. M. VERNON
M.A., M.D.



WE REGRET TO record the death on February 11, 1951, of Dr. H. M. Vernon at the age of 80 years.

Born on October 3, 1870, Horace Middleton Vernon was educated at Dulwich College. Thence, in 1888, he proceeded to Merton College, Oxford, where for five years he studied chemistry and physiology. At that time he was interested in general biology, and after 1893 he spent much time doing biological research at Naples. Between spells at Naples he studied medicine at St. George's Hospital, and he qualified in 1896.

From 1898 to 1920 he was a Fellow of Magdalen College, Oxford, and he was also University Lecturer in chemical physiology.

Up to the first world war he continued with teaching and research in physiology against the quiet background of Oxford. His interest in industrial conditions was aroused in the summer of 1915 when he volunteered for work at the bench in a Birmingham factory. A vacation spent in gauging shells, with a nominal working week of 74½ hours and much overtime, introduced him to a field of experience of which he had previously been ignorant.

When, later in 1915, the Health of Munition Workers Committee was set up, mainly because of the toll that work for inordinately long hours was exacting in the form of reduced output and increased sickness, Vernon offered his services. He still had his teaching obligations, but, as they permitted, for the rest of the war he carried out his investigations in munition factories. His extensive studies of the effects of hours of work and arrangement of shifts on output, safety, and sickness yielded results of the utmost economic as well as scientific value. During these investigations he showed the important effects of temperature on accident liability. At this time he was also persuaded to study the influence of alcohol on working efficiency.

In 1918 the Industrial Fatigue (now Health) Research Board was created to continue the work of the wartime Committee, and Vernon was invited to take up a full-time appointment. At the cost of relinquishing his Oxford appointments he did so, for he was much attracted by the opportunity for social service that industrial health research offered. Towards the end of his life he wrote that he had never regretted this decision.

His researches since the first world war will be familiar to many readers of this journal. In the early years he carried out researches on fatigue and efficiency in the tinplate and iron and steel industries and in the potteries. Then, after studies of the effects of rest pauses and hours of work he turned his attention to studies of heating and ventilation and the effects of the thermal environment on efficiency and well-being. There was an interlude in which laboratory studies of static effort were made.

He retired from active work in 1932, but for several years thereafter he occupied himself in writing books on some of the subjects on which he had earlier done active research. He was keenly disappointed when in 1940, in the second world war, the lessons of the earlier war concerning the evils of excessive hours of work were almost ignored, and he did what lay in his power to draw attention to the mistake. He produced a book on

the health of munition workers, and wrote several articles for periodicals.

Vernon was one of the outstanding pioneers in industrial medicine. During the 17 years or so for which he was engaged in active research he made many major contributions to our knowledge of man's reactions to his hours and conditions of work which will be of lasting value. His earlier papers, published as memoranda of the Health of Munition Workers Committee or as reports of the Industrial Fatigue Research Board, contain masses of data from which students in future years will profit.

THOMAS BEDFORD

BOOK REVIEWS

Appraising Vocational Fitness by Means of Vocational Tests. By Donald E. Super. 1949. New York: Harper. Pp. xxi + 727. Price 44*s*.

The Structure of Human Abilities. By Philip Vernon. 1950. London: Methuen. Pp. 160. Price 12*s*. 6*d*.

There is considerable ignorance about the value of psychological tests in industry because of the absence of reliable sources of reference which are truly critical without being incomprehensibly technical. *Appraising Vocational Fitness* meets this need to a considerable extent, in spite of its necessarily American background so far as specific tests are concerned. Dr. Super, an Oxford graduate who is now a professor of education in the Department of Guidance, Teachers College, Columbia University, has set out in his 24 chapters and two valuable appendices a wealth of wisdom and fact. The 995-item bibliography attests both to the volume of research and to the author's breadth of scope in this work. For those who feel a need for what may be called the philosophy and method of psychological testing for occupational purposes, this book will be worth reading, even if the discussion of 42 well-known tests is never consulted. This may best be illustrated by means of a few extracts, for example (p. 6):

> "In order to evaluate a person's vocational prospects, two types of information about him are needed: the *psychological* facts which describe his aptitudes, skills, interests, and personality traits; and the *social* facts which describe the environment in which he lives, the influences which are affecting him, and the resources which he has at his disposal . . . . it seems desirable, in considering the types of data needed in vocational diagnosis, to stress the need to obtain both types of information and to use both testing and non-testing techniques."

This comes in welcome contrast to the excesses of the "testophile" and "testophobe", as does a further passage on page 8:

> "Despite the great progress in psychological testing since World War I, the variety of characteristics which can be measured still leaves a great deal to be desired. As is made clear in greater detail in subsequent chapters, the measuring instruments we now use even for the most adequately measured traits, such as intelligence and vocational interest, are still crude and only half-understood; those we use for measuring personality traits, such as general adjustment, introversion and the need for recognition, are still in embryonic stages; and there are no methods of testing creative imagination, persistence, and certain other traits and abilities which are often assumed to be important and which laboratory studies and other types of investigations have suggested may actually exist. For these reasons the psychological study of a person's abilities and personality traits requires more than testing techniques. When a suitable test is available, its use will generally save time and obtain the information in a more objective, valid, and usable form than would otherwise be the case. This is especially true of intelligence, and it applies also to a variety of other traits. But some tests measure aspects of ability or interest which are so narrow as to make their use dangerously misleading unless the data obtained with them are thought of as being only one small part of the aptitude picture . . . ".

This section leads on to a brief but useful examination of such familiar non-test techniques as the interview, questionnaires, and rating scales. While the value of each is fully recognized by Professor Super he does quote damaging objective evidence of their weaknesses and lack of validity in a variety of situations. He distinguishes clearly between the uses of tests for *selection* and for *vocational guidance*, and devotes much-needed attention to the neglected subject of *criteria for occupational success*.

> "Educators and business men who are not trained in statistics and in experimental methods, and some who are trained in experimentation in other fields but not in psychology, often fail to realise that in a blanket questioning of the validity of tests they assume the validity of some other criterion or predictor such as school marks, supervisors' ratings, production records, or their own judgement. They too often do not know how unreliable or invalid these other indices have been shown to be by objective investigations" (pp. 12–13).

His warnings about the consequences of careless administration of standardized tests are most timely; in the reviewer's experience, such disregard of directions and procedure is widespread.

The brief introduction to basic statistical concepts, without which objective evaluation of appraisal methods is impossible, is admirably lucid and justifies the author's remark that "statistics is nothing more than logic expressed in numerical form: therefore any reader who can engage in logical reasoning can master elementary statistics, and those who enjoy logic should enjoy statistics" (p. 643). He makes some trenchant comments about the widespread misuse of the term "prediction" as applied to selection and guidance; for example (p. 661):

> "The situation would be improved if the general public, and some psychologists whose absorption in test construction has caused them to lose sight of the context in which they work, would cease to think in terms of predicting the success or failure of individuals, and come to think in terms of probabilities, some of the contingent factors of which will remain unknown even after the most thorough of testing and interviewing procedures."

Although this book is designed primarily for specialists in personnel selection and vocational guidance, it can be recommended also to those who, while not specialists in this field, are often expected to be by virtue of their role and status in industry.

Professor Vernon's book, *The Structure of Human Abilities*, the first of a new series entitled "Methuen's Manuals of Modern Psychology", is intended by its author to provide in a simple, non-technical form that "acquaintance with the principles and results of factor analysis necessary for the study and practice of educational, vocational, or other branches of applied psychology". There will be many readers of this journal who do not feel any great stirrings within them to make the acquaintance of factor analysis. For them the attraction of this book by the Professor of Educational Psychology in the University of London is more likely to lie in his second purpose :

> "To bring together the large number of publications in this field, in Britain and America, which at first sight appear to give contradictory and confusing accounts of mental structure, and to show that they can be fitted into one consistent—even if incomplete—picture."

He has achieved a considerable success in both these aims. Like Professor Super, he realizes that any mention of mathematics tends to "frighten or antagonise many of the teachers, employers, and others who are most prone to discuss abilities unscientifically", but goes on boldly to demonstrate very convincingly that the basic principles of factor analysis are really quite simple to those who have not made up their minds in advance that they couldn't possibly grasp them. The nature of such mathematically derived factors in psychology is stated clearly as "categories for classifying mental or behavioural performances, rather than as entities in the mind or nervous system", and the author is consistent in opposing the assumptions that underly the "faculty" approach of many professionally qualified people to the subject of human abilities. The book is committed to the "hierarchical" theory which has developed steadily in this country during the past 40 years, giving prime importance to "general mental ability" (Spearman's "g") and then to two well-marked group factors styled "v : ed." (verbal : educational) and "k : m" (spatial : mechanical). He adopts a pragmatic criterion for the general acceptance of further major group factors at this level, namely that they shall be shown to have importance in their application to educational, occupational or other fields in daily life. In this he typifies the British position in contrast to that of the Americans, and in the process makes it clear that the latter are already showing some signs of recognizing the points of contact revealed even while adhering to the Thurstone "multiple factor" approach popular there. Apart from examples in the text, this receives lucid treatment in a short appendix, and the 265-item bibliography should provide adequate source material for those who wish to follow-up in detail any special aspect of human abilities. It is a little surprising to find on the last page of the Appendix a defence of "the I.Q., or other comparable measures for adult purposes" which does not more fully indicate the great importance now attached to the "other comparable measures". Although Professor Vernon is concerned to show the relation between the hierarchical theory of human abilities and the satisfactory nature of reliable tests yielding a global score which can be expressed in the form of an intelligence quotient, no risks should be taken which may result in the perpetuation of the use of I.Q. measures for *adult* intelligence which are derived from the "mental age" concept familiar from the Stanford-Binet scale. Another criticism is the absence of an overall summary in which the conclusions of various chapters can be brought together. An important summarizing paragraph occurs on page 86, at the end of Chapter VII, which is entitled "Practice, Difficulty, Speed and Other Factors"—not a place where one would expect to find it—but the main text ends on page 128 with only the usual chapter summary. This is not an easy book, but that is because understanding of the mental functions cannot be made easy. Professor Vernon's book constitutes an important step in the process of making available to the layman the results of immense quantities of research into the nature of human abilities as particularly related to educational and occupational problems.

These two books, one American and the other British, give some indication of the rapid development of the theoretical, methodological, and practical aspects of one of the younger branches of scientific enquiry. Both authors are rightly modest about the limits of present knowledge ; both are aware of the dangers of over-enthusiasm ; both are explicit that users of psychological methods and assessments must accept rigorous standards of administration and evaluation ; and both expect the intelligent cooperation of their non-specialist readers to the extent of mastering simple mathematical and logical concepts, and of the adoption of an unprejudiced attitude towards their subject matter. In the reviewer's opinion, both have rendered a service to many, such as those engaged in industrial medicine, who have recognized the importance in their daily work and relationships of having some reliable information about these matters.

ALASTAIR HERON

Industrial Hazards. British Medical Bulletin, Volume 7. No. 1–2, 1950. London : Medical Department, The British Council. 40 illustrations. Pp. 1–79. Price 10*s*.

It is useful to pause and take stock of the situation, to look back on what has been done, and to see clearly the tasks that lie ahead. To keep up to date with advances in industrial medicine is a formidable problem. We therefore owe a debt to the British Council for gathering a symposium, which brings us up to date with the present state of knowledge concerning industrial hazards. It does not pretend to be an exhaustive account of every aspect of industrial medicine ; skin and bladder cancer, for example, are not mentioned, although readers of this Bulletin will remember a previous number devoted to "Chemical Carcinogenesis", but its scope is sufficiently wide for Dr. Rogan to say in his preface : "It is unlikely that the doctor who spends all or part of his time in industry will encounter the

majority of the hazards described here ". Our individual horizons tend to be limited, but it is necessary that we should be aware of the clinical, pathological and preventive aspects of all the hazards in the wide field of industrial medicine.

It is difficult to do justice to the contributors in a short review, particularly as they have each produced interesting accounts of their own particular subjects. Little more can be done than to give a very broad outline of the contents of this instructive series of papers.

Dr. Donald Hunter discusses the toxic metals, omitting those already familiar. In addition to a timely survey of beryllium he reviews the literature of cadmium, osmium, platinum, selenium, tellurium, uranium, and vanadium. Chrome and manganese, well known in the literature, have come into increased use of recent years, and their effects are also described. He draws attention to the importance of substituting harmless halophosphates for beryllium salts in the manufacture of fluorescent lamps, and says that these halophosphates *must be used*.

Dr. Taylor deals with the toxic effects of some new compounds not in common use but likely to become of great importance, among them the silicones and the fluorinated paraffins. His paper illustrates the great value that the chemist can be to the industrial hygienist in elucidating toxic hazards before substances are brought into general use.

Dr. Browning, whose new edition of " Toxicology of Industrial Solvents " is still awaited, reviews the properties, industrial uses, and toxicology of the aromatic and cyclic hydrocarbons, impressing on us once again the dangers of benzene. Too much cannot be said about the toxic effect of this common solvent. Many chemists would do well to remember the recent dictum of a chemical works manager : " Benzene is not a hexagonal solid, it is a colourless liquid which gives off a dangerous vapour ".

The new organic insecticides have been much in the news, and it has been shown that many of them are as toxic to man as they are to the insects. Dr. Barnes describes them and points out that, although the acute manifestations are becoming well known, the effect of absorption of small doses over a long period is neither easy to estimate in experimental animals, or to predict in human beings. As we cannot prevent their use, we must be prepared with methods to eliminate their undesirable effects. These are particularly difficult to apply without effective legislation.

The paper on occupational skin diseases by Squire, Cruickshank, and Topley is an excellent piece of fundamental research. They approach this vexatious problem from the statistical, clinical, and bacteriological angles and, having shown from national and local statistics how the incidence of dermatitis has been rising, they discover in the course of a field investigation the substances responsible for dermatitis in the light engineering industry. Measures for prevention and treatment, based on clinical and bacteriological findings, are suggested, but it is emphasized that there is still need for further research.

Respiratory disease is the subject of the next three papers. Fletcher and Gough describe their work on coal miner's pneumoconiosis, illustrating their article with some excellent pictures of Dr. Gough's whole-lung sections. Dr. Middleton, whose international reputation is well known, gives a short, concise, yet easily assimilable account of silicosis and other pneumoconioses. His ease of presentation of this wide subject is a tribute to his long experience. Fresh interest has recently been aroused in the problem of byssinosis in the cotton industry, and Dr. Schilling gives an account of some of the work done recently by the Department of Occupational Health at Manchester University. He clears up the confusion which has existed in the literature between " mill fever " and " Monday fever ", and draws attention to the new method of oiling cotton which bids fair to remove much of the trouble arising in cotton card rooms.

Dr. Edson reviews the occupational radiation hazards. After listing the occupations which present these hazards he shows what clinical results have appeared from exposure, and, having pointed out the methods of evaluating radiation hazards and measuring doses, he discusses radiation protection, pointing out that health supervision, although essential, is no substitute for the proper planning of methods and training of operatives. As a depressing corollary to Edson's paper, Dr. Loutit discusses the treatment of the radiation syndrome. Although positive information is scanty, efforts are still being made to discover methods of preventing the ill-effects of over-exposure to radiations, and certain lines of research appear hopeful.

The last three papers in the symposium concern industrial injuries. Dr. Bull analyses and classifies them, pointing out from his experience that probably only one-seventh of the total injuries received are actually treated at the works surgery. He illustrates the proportions of incidence and of lost time due to compensation accidents, and analyses the fatal industrial accidents in the Birmingham area. Apart from the prevention of specific injuries, he points out that recent advances in treatment, such as the closure of major open wounds and chemotherapy, could be used more extensively. Whitfield stresses the importance of record keeping. He admits that although it is possible to do much by the design of equipment, and by paying attention to lighting, temperature, humidity, and hours of work, there remains the problem of individual emotional and social conditions which are difficult to assess, and play a large part in the cause of accidents. The prevention of sepsis in minor industrial wounds is discussed by Dr. Williams, who has done so much to elucidate this problem. A great deal can be done by organization in the factory surgery and using a " non-touch " technique, but there are two problems which remain unsolved : (1) the prevention of the trivial wounds which lead to bacterial invasion, and (2) some method of treatment capable of being used in the many factories which have no adequately trained first-aid workers.

The plates illustrating the effects of beryllium, chrome, industrial dermatitis, coal miner's pneumoconiosis, and fullers' earth pneumoconiosis are well produced, but the photographs in the text leave much to be desired. This is a minor criticism, and does not detract from the conclusion that this work is a really important contri-

bution to industrial medicine. It is worth every penny of ten shillings. R. MURRAY

Blindness : Modern Approaches to the Unseen Environment. Edited by Paul A. Zahl. 1950. Princeton University Press (London : Geoffrey Cumberlege). 22 plates. Pp. 576. Price 48*s*. net. ($7.50).

This book, the combined work of 31 contributors, most of whom are experts in some particular field of blind welfare, is the most comprehensive publication on the problem of the blind which has yet been written in the English language. The word "problem" is used advisedly because, although all the contributors are obvious enthusiasts and have much to say on what has been achieved, one is struck perhaps most forcibly by the emphasis they place on how much there is still to be learned of the psychology of the blind before his problem can be truly solved, on how far from their goal even the most highly organized schemes still are, and how still imperfect are the many devices which have been invented to aid the blind. The other striking general impression which the book gives is how far removed from benevolent but sterile patronage is modern blind welfare ; with all its ramifications its single purpose is to make the blind self-respecting and self-supporting.

The book is divided into seven parts. The first is concerned with the history of modern work for the blind from its foundation by Valentin Haüy in Paris in 1785, when he started the first school for the blind, to the complexity of agencies, associations, societies, schools, schemes and legislative acts which operate today in the United States, Great Britain, Canada, and continental Europe. A distinct difference is remarked upon in policy on the two sides of the Atlantic. In America this is based on the principle of, as far as possible, educating, rehabilitating and training the blind at or from his own home, whereas in Great Britain and Europe the preference is for residential institutions.

The next two parts cover education, psychology, rehabilitation and training in general, separate sections dealing with the pre-school child, schooling, the adult, the aged and deaf blind. If these 11 chapters with their multiplicity of agencies, organizations, schemes and acts, with a leavening of psychological terms and somewhat burdened by repetition make rather heavy going for the amateur, they cannot fail to bring home to him the magnitude of the problem and are certain to be of great value to the expert.

After a section on the military blind, which includes a concise review of St. Dunstans by Sir Ian Fraser, the last half of the book deals in detail with aids to the blind. It is both easy to read and of absorbing interest. First are discussed the time-tested aids such as Braille and its rivals (there is an interesting account of its history and of the "battle of the types"), the talking book, the white cane, proficiency with which requires quite an elaborate technique, and the guide dog. Mention is made of aids to cooking, sewing, various games, and a number of manual trades and other activities. There are three chapters on aids which are in process of development, such as the recording machine, and on methods of research into the efficiency of existing and experimental devices ; two further chapters describe reading machines and their future potentialities and the possibility of employing phosphene phenomena in the production of artificial sight. A final chapter summarizes the causes of blindness.

With the qualification that 90% of the publication is American in subject matter and in method of presentation, it is certain to be of interest and value to anyone connected in any way with the welfare of the blind.
A. LISTER

Eyes in Industry. By H. S. Kuhn, M.D. 1950. 2nd Edition. London : Henry Kimpton. Pp. 378. Price 60*s*.

Those of us who had to deal with eye problems in industry during the war years were always grateful for the help and guidance given in the first edition of this book. At that time there was practically no literature dealing with these problems.

This new edition has been enlarged, and like the majority of American publications is well printed and profusely illustrated with a series of excellent and instructive photographs. The advice given throughout, both on methods of prevention and on treatment, is sound, but one wonders to what extent the various eye programmes described are found in industry in the United States.

While no one could possibly cavil at the necessity for an adequate pre-employment ophthalmic examination, yet one does wonder what may be the ultimate result of some of the complete screening procedures of all industrial employees. In many industries a strict interpretation of desirable visual standards for those already in employment might well lead to almost insoluble problems in job placement. While periodical examination is undoubtedly essential in certain specific instances, such as crane drivers and locomotive drivers, for the great majority of workers, the examination is possibly better confined at the present time to improving their visual capacity, rather than in finding them unsuitable, judged by an arbitrary standard, for an occupation which they may have had for many years.

In many British industries there is undoubtedly room for improvement in the methods of conducting the initial eye examination, which is still too often done in an unsatisfactory and slipshod manner, but the question of suitable visual standards in industry is still one which has to be settled arbitrarily, and is influenced, not only by the type of industry, but by the availability of labour. Many of us have experienced difficulty in assessing the question of depth perception, and it is interesting to find that even in the United States they have not yet developed an entirely satisfactory method for investigating this faculty.

In Great Britain, where there is at present full employment, the difficulty is not so much one of selection from a number of suitable persons, as one of making the best of the material available, and indeed of thinking more in terms of minimum visual standards.

The chapters dealing with treatment are undoubtedly rather too detailed for the average industrial medical officer, and perhaps more emphasis might be laid upon

the advantages of the routine use of penicillin preparations after the removal of foreign bodies.

It is interesting to see that speed in irrigating chemical eye injuries is emphasized, as this is still too often overlooked; but there does not appear to be any mention of the use of the buffered phosphate solutions which are now used in Britain as universal antidotes.

There is an interesting chapter on epidemic keratoconjunctivitis which was a serious problem during the war years in America, and from which we in this country were fortunately spared.

While the phraseology and wording of this book is somewhat unusual and confirms its American origin, it undoubtedly contains a considerable amount of interesting material, and could well be included in the library of every industrial medical officer.

H. F. CHARD

Medical Aspects of Atomic Warfare. *The Practitioner* (December, 1950). No. 990, Vol. **165.**

This issue of *The Practitioner* consists mainly of a symposium upon the "Medical Aspects of Atomic Warfare" prepared with the help of Sir Ernest Rock Carling. Ten papers are provided covering a wide field without undue recourse to the complexities of atomic physics or radiobiology.

Sir Ernest Rock Carling's introductory article "Morale: an Exercise in Preventive Medicine", places emphasis upon prior education, information, discipline, and an effective medical service in maintaining national morale during atomic warfare, thus providing one of the most effective antidotes to the weapon's effects.

C. G. Neary ("A Simplified Account of Atomic Radiation Physics") prefaces his account by emphasizing that the most dangerous potentialities of atomic explosions are the thermal and blast effects; the radiation effects, while "novel", are not first in order of importance. He explains in a reasonably straightforward fashion such elements of atomic physics as are required to appreciate the problems likely to arise from different methods of atomic attack.

R. H. Mole ("The Action of Radiation on Tissues") gives a broad outline of the physiological and pathological sequelae of irradiation, and surveys the possible mechanisms of acute and delayed radiation injuries.

Brigadier A. Sachs ("Morbid Anatomy of Radiation Injuries") summarizes the pathological findings in the Japanese victims of 1945, and indicates broad clinical groupings of radiation casualties.

D. G. Catcheside ("Radiations and Genetics") discusses briefly the essentials of normal genetics, the process of mutation and some of its potentially adverse consequences in man. He then considers the mutagenic effects of radiation in animals and plants, and the evidence on the eventual genetic effect of irradiating a proportion of the country's population. His main conclusion is that there is likely to be a long-term genetic result from the irradiation received by surviving persons, although he is naturally guarded about its extent or significance.

E. Leader-Williams and J. Smith ("Assessment of Possible Casualties and Damage") give a sober and sobering evaluation of the probabilities of the effectiveness of atom-bomb attack, based on an analysis of the results of the 1939–45 war's H.E. bombing attacks, and the Japanese atom-bomb data. This article, perhaps above all others, draws attention to the probable magnitude of the destructive, social, and medical sequelae of atomic explosions in such a country as this, and points clearly to the most urgent problems of defence and medical organization.

Sir Claude Frankau ("The Casualty Service") considers the methods by which the nation's civil defence and medical services could best cater for the initial and secondary problems from atom-bomb attack, with due emphasis upon the massive scale of preparedness required to deal with the potential needs.

J. F. Loutit and W. d'A. Maycock ("The Treatment of Radiation Casualties"), after drawing attention to the probability of a higher incidence of casualties from non-radiation rather than radiation injuries, summarize the likely clinical courses of acute radiation injuries, and discuss nursing and therapeutic measures for casualties. Once more, it seems that the best lines of treatment as yet available concentrate mainly on good nursing, the prevention of concurrent infections, and the transfusion of blood. Although many pharmacological avenues have been explored, there seems up to now no effective therapy for the acute radiation syndrome.

J. P. Bull ("The Treatment of Flash-burns") summarizes present-day concepts of the treatment of burns affecting relatively large areas of the body surface. As in other articles, the probable need for urgent treatment of very large numbers of cases of burns within a short time is emphasised and methods for simplifying the initial and plenary treatments of the burned cases are suggested.

A. E. Martin's article ("Monitoring Instruments and their Use in Civil Defence") is perhaps a little short and comes rather late in the symposium. This may tend to imply that the use of monitoring instruments for dose-measurement and contamination-detection is somewhat of a side-line to the medical matters described in preceding papers. That is unlikely, since a knowledge of the order of radiation exposure received by a casualty will lead to better patient-grouping and disposal, better casualty reception and availability of treatment than would occur were all cases to be received and disposed of without regard to their magnitude of exposure. It would have been better if this paper were made to follow Neary's paper on the physical aspects of the explosions, and enlarged to conform to the real status of instrumentation during and after radiation emergencies.

The Practitioner has provided a helpful collection of expert opinion and knowledge on an important subject. As in most symposia, there is some repetition, but this—often annoying—occurrence is for once somewhat helpful, for the medical aspects of atomic warfare are not always easy to understand, especially where they involve the physical aspects of atomic explosions. The symposium provides the general practitioner or the industrial medical officer with an amount of information which is probably adequate for all present purposes.

There is of course no orthodox industrial medicine

in the symposium, and it would be difficult to regard the subject as one of occupational health. Nevertheless, the papers presented are of rather special value to the industrial medical officer and should help him to give a reasonably assured, knowledgeable, and unhysterical view of the medical aspects of future atom-bomb attack. And in this respect the medical officer is always regarded as "one who should know". His experiences with hazard evaluation, casualty prevention, first aid, casualty disposal, group discipline and group morale can be of high value in such emergencies. E. F. EDSON

The Industrial Safety Manual and Directory. Edited by J. Vernon Bosly, B.Comm. 1950. London : Practical Press, Ltd. Illustrated. Pp. 206. Price 25*s*. net.

In this book the Editor contributes a well balanced survey of the steps which should be taken to reduce hazards to health and safety ; he discusses protective equipment, good housekeeping, and the education and training of management and workers. The provisions of the Factories Act in respect of accident prevention are set out in readable form, and the constitution and functions of a safety committee are described. There is a good account of the nature of mechanical hazards and the best ways of reducing them : common faults in electrical installations are illustrated by simple diagrams, and other chapters deal with dangerous chemicals, dust control, the risks of fire, and with ventilation, heating, and lighting. An interesting article points out the value of colour in the industrial environment ; suitable colours are suggested for reducing the chance of accident in handling machines and in getting about.

Particulars are given of the principal organizations concerned with occupational health and welfare, and there is a useful list of periodicals and books on these subjects.

Finally, the book contains an alphabetical list of safety equipment and protective devices, which is cross-indexed with a list of firms and other agencies which supply these articles.

The Editor is to be congratulated on the production of this book, which is likely to be most helpful to all who are seeking to raise the standards of health and safety in industry. D. C. NORRIS

Safety in the Chemical Laboratory. By H. A. J. Pieters and J. W. Creyghton. 1951. London : Butterworth & Co. (Publishers) Ltd. Pp. 258. Price 15*s*.

This is a deadly serious book translated from the Dutch. It is intended to be at the elbow of every technician to warn him of the risks of his job and to tell him how to work safely. It is doubtful if any book can replace or even aid the learning from precept and example that forms the basis of technical skill.

The style of this book is jerky, disordered, and repetitious. It is characterized by a mixture of pomposity ("a fire is an exothermic reaction, mostly between an oxidizable substance and oxygen") and of naivety ("special care should be taken in the storage and transport of glassware and chemicals"). The book owes its length to its repetitiveness and to the inclusion of much irrelevant material that has replaced the detailed consideration of safety measures that should be the main object. Thus the treatment of cuts and wounds is given three lines whereas methods for dust sampling occupy seven pages : radiation hazards in the modern chemical laboratory receive 15 unhelpful lines and the methods for the estimation of carbon monoxide in the air and in blood, 17 pages. The methods for the detection of alcohol in the breath, blood, and urine (pages 180–184) also seem out of place in a book on safety.

Quite apart from defects in style and proportion the book fails chiefly because it is an inadequate guide to the trivial as well as to the major problems in laboratory management. Such statements as, "Handling a pipette frequently causes accidents especially with iodine or alkali hydroxide solutions. A safety pipette or a pump should be used", on page 51 and repeated in essence on page 124, make up about a third of the book. If the technician is so inexperienced that he is unaware of these points he will not be helped much by this exhortation. If the advice is considered necessary it should be amplified by a description of the safety pipette and the way in which the pump is used. As an example of omission on a larger scale, the problem of chemical carcinogens can be considered. Chemical carcinogens are discussed briefly under the heading "Toxicity and Chemical Constitution", and a page is given to the structural formulae of seven of these. This space could have been put to better use by giving an account of the types of laboratory in which risk of exposure to carcinogens is known to occur, the precautions that are taken in such laboratories, and the supervision of personnel exposed to this particular risk. But detailed information of this sort is not to be found in this book.

F. A. DENZ

PROCEEDINGS OF THE ASSOCIATION OF INDUSTRIAL MEDICAL OFFICERS

61st Annual General Meeting

The 61st annual general meeting of the Association was held in the Lecture Theatre of the Royal Society of Tropical Medicine in London, on October 27 and at the London Hospital on October 28, 1950.

On the morning of October 27 the following papers were read: "Preventive Medicine in Weed and Pest Control" by Dr. A. David, medical officer, Pest Control Ltd.; "The Work of a University Department of Industrial Health" by Professor R. C. Browne, Department of Industrial Health, King's College, Newcastle-upon-Tyne; "Some Problems of Railway Medicine" by Dr. G. O. Hughes, medical officer, British Railways; "Some Human Relation Problems in Industry" by Dr. D. S. F. Robertson, medical officer, Lever Bros. and Unilever Ltd.

This session was followed by a symposium on "Accident Prevention", in which the following speakers took part: Mr. G. P. Barnett, H.M. Chief Inspector of Factories, Ministry of Labour and National Service, who spoke on "The Fundamentals of Accident Prevention"; and Dr. J. A. A. Mekelburg, medical officer of Peek Frean and Co. Ltd., on "Industrial Accidents—a National Problem". The symposium was concluded by Dr. T. Bedford, Director of the Environmental Research Unit, Medical Research Council, who spoke on the subject of "Thermal Conditions in Relation to Accidents".

In the evening a dinner was held at St. Ermin's Hotel, London, when an address was given by Mrs. Helena Normanton, K.C. entitled "Doctors as Criminals".

On October 28, 1950, a clinical meeting and demonstration was given by Dr. Kenneth Perry at the London Hospital. The subject was "Medical Cases from the Point of View of Industry".

Branch Meetings

Since the last issue of this journal the following reports have been received.

The **Tees-side Group** heard at their November meeting an address by Dr. Williams, consultant physician, Middlesbrough General Hospital, on "A.C.T.H. in Relation to the Adaptation Theory".

The third annual business meeting of the **South Wales and West of England Group** was held in October, when the Chairman, Dr. J. Gwynne Morgan, and Secretary, Dr. T. H. Jenkins, were re-elected.

A visit was paid to the Landore Works to see a new silica brick works and a new ingot mould works being constructed, to the National Dock Labour Board port medical services in Swansea, and to the factory of the Viscose Development Company.

The **Scottish Group** met in Glasgow in October as the guests of the Industrial Nurses' Group, when an address was given by Miss Carol Mann, industrial nurses' organizer of the Royal College of Nursing, on "Nursing Aspects of an Occupational Health Service".

For the November meeting a whole-day visit was paid to the Ardeer Factory of I.C.I. Ltd.

A meeting of the Yorkshire Group was held in Sheffield in October jointly with the **North Western Group.** Members visited the new medical department of Messrs. Steel, Peach, and Tozer as guests of the medical officer, Dr. R. A. Trevethick. The clinical session was devoted to a showing and discussion of the B.M.A. film "The Treatment of Infections of the Hand".

The **North Western Group** held a three-day meeting at Whitehaven in September, when the programme included visits to the haematite mines at Beckermet and Egremont and to the clinic for the mine workers at Whitehaven, where Dr. Craw, the medical director, outlined the development of preventive measures from 1935 till the present day.

The September meeting of the **Birmingham and District Group** took the form of a visit to the works of Needle Industries Ltd., Studley, which has 400 employees and a weekly output of 30 million needles. The annual business meeting was held in October, when Dr. N. G. Marr was elected chairman and Dr. J. G. Lawson representative on the Council of the Association. After the business meeting Dr. Dorothy Campbell spoke on "Miner's Nystagmus".

The November meeting was a joint one with the Industrial Nurses' Group of the Birmingham and Three Counties Branch, Royal College of Nursing, when the following papers were read: "Backache" by Dr. A. Hutton Wilson; "Dust Diseases of the Lung", by Dr. A. J. Amor; "The Placement in Industry of the Tuberculous Worker" and "Recent Advances in the Treatment of Tuberculosis", by Dr. J. E. Geddes; "General Medical Practice in Relation to an Industrial Medical Service", by Dr. R. C. L. Burgess and Dr. R. J. F. H. Pinsent.

In December a visit was paid to the Netherton Iron Works, Dudley.

ABSTRACTS

(This section of the JOURNAL *is published in collaboration with the two abstracting Journals,* Abstracts of World Medicine, *and* Abstracts of World Surgery, Obstetrics, and Gynaecology, *published by the British Medical Association. The abstracts are divided into the following sections : toxicology ; industrial physiology ; industrial lung disease ; industrial skin diseases ; accidents and orthopaedic surgery ; industrial ophthalmology ; environment ; general. Not all sections will necessarily be represented in any one issue)*

INDUSTRIAL TOXICOLOGY

Poisoning by Organic Phosphorus Insecticides. BIDSTRUP, P. L. (1950). *Brit. med. J.*, **2**, 548.

Several alkylpolyphosphates have recently been introduced into agriculture as insecticides, especially against aphis, red spider, and other species which may be resistant to DDT. Unfortunately, little is known as to their acute toxicity for man and even less as to the possible effects of repeated absorption of minute amounts. Hexaethyl - tetraphosphate (HETP), tetraethyl - pyrophosphate (TEPP), and diethyl - *p* - nitrophenyl - thio - phosphate (" parathion ") are now used quite widely, though the last seems to be supplanting the other two owing to its slow rate of hydrolysis and consequently longer action. All these substances act like di-isopropyl fluorophosphate (DFP) as inhibitors of cholin-esterase. With the first two compounds 16 cases of poisoning, with no deaths, have been reported from the U.S.A., while more than 100 cases, with at least 6 deaths, have occurred in one spraying season with parathion.

Cholinesterase inhibition is due to its combination with the inhibiting agent. With DFP the combination is irreversible almost at once, and recovery depends on the possibility of regeneration of the esterase. With the others, the irreversible stage is reached more slowly and hence the immediate administration of atropine in adequate dosage (1 to 2 mg. repeated hourly till the pupils dilate or the signs of excess parasympathetic action have disappeared may be successful. The lethal dose of parathion for man is not accurately known, but is believed to be very low—perhaps less than 10 mg. Very little is known as to the amounts required to cause chronic poisoning.

Parathion can be absorbed through the skin, the lungs, or the alimentary tract. The earliest symptoms are usually anorexia and nausea, made worse by smoking or eating, soon followed by vomiting, intense colic, sweating, and salivation. The pupils are constricted in moderately severe poisoning, but may be normal in slight cases. All these signs and symptoms are evidence of excessive parasympathetic stimulation. In addition there are twitchings of the muscles, occurring early and first seen in the eyelids and tongue. In severe cases, these may spread to the face and neck and even become generalized. Neuromuscular paralysis may occur, with death from respiratory failure. Atropine does not antagonize acetylcholine at the myoneural terminals in striated muscle and hence is ineffective in such cases, so that provision should be made for artificial respiration to be started immediately neuromuscular paralysis develops. Other signs of excess parasympathetic action—diarrhoea, involuntary defaecation, pulmonary oedema, etc.—appear in acutely ill persons. Affection of the central nervous system shows itself in giddiness and apprehension, followed by headache, ataxia, and drowsiness. Coma sets in gradually, with loss of reflexes but with generalized convulsions. Death occurs within 24 hours, at an average of about 9 hours from the last exposure.

Preventive measures entail provision of overalls fastening closely at the neck and wrists, gloves, boots, and aprons. A full-face gas-mask alone can protect from inhalation dangers. No one who has been affected should be allowed to work with these substances until his cholinesterase activity has returned to normal.

Reginald St. A. Heathcote.

Studies on the Toxicity and Pharmacological Action of Octamethyl Pyrophosphoramide (" Ompa " ; " Prestox III "). DUBOIS, K. P., DOULL, J., and COON, J. M. (1950). *J. Pharmacol.*, **99**, 376.

Octamethylpyrophosphoramide (" OMPA ") is a systemic insecticide and is also toxic to mammals. The toxic and pharmacological actions of this substance were studied. Acute toxicity was measured in rats, mice, guinea-pigs, and dogs. LD 50 values (in mg. per kg.) were : rats 8, mice, 17, guinea-pigs 10 (intraperitoneally) ; dogs 5 to 10 (intravenously). In rats the oral LD 50 was 10. In all these species the symptoms were typical of *para*sympathomimetic drugs except that those referable to stimulation of the central nervous system were absent.

Large intravenous doses in dogs caused an immediate evanescent but non-specific fall in blood pressure and increase in respiratory rate, followed by bradycardia and increase in pulse pressure culminating in cardiac failure.

OMPA was ineffective as an anticholinesterase agent *in vitro*. However, 2 hours after intraperitoneal injection of 5 mg. per kg. into rats, the following percentage

inhibition of cholinesterase activity was observed in various tissues : submaxillary tissue 89, ileum 70, serum 87, skeletal muscle 68, and brain 2. After lethal doses, the percentage inhibition in peripheral tissues was 90 but in brain only 7. The *in vivo* inhibition is reversible. OMPA is changed by the liver into a strong anticholinesterase agent. Plants grown in soil containing OMPA contain a similar agent.

In mice, atropine premedication increased the LD 50 four-fold and atropine-eserine increased it eleven-fold. Eserine itself had little prophylactic effect. Atropine therapy in dogs increased the LD 50 about four-fold.

Vera N. Warren.

Acute Inhalation Toxicity of Beryllium. 1. Four Definitive Studies of Beryllium Sulfate at Exposure Concentrations of 100, 50, 10 and 1 mg. per Cubic Meter. STOKINGER, H. E., SPRAGUE, G. F., HALL, R. H., ASHENBURG, N. J., SCOTT, J. K., and STEADMAN, L. T. (1950). *Arch. industr. Hyg. occup. Med.*, **1**, 379.

Experimental production of the acute response to inhalation of beryllium compounds is described. Methods are described in detail for the production of a mist of beryllium sulphate in concentrations between 100 and 1 mg. per cubic metre of air. A total of 11 species of animals were exposed, repeated daily 6-hour exposures being lethal to most species at concentrations of 50 mg. per cubic metre and above.

There were two types of response : an acute lethal phase with few histological changes, and a delayed phase in which surviving members on further exposure appear at first to suffer little effect, but show increasingly severe changes on continued exposure. Details are given of changes in the body weight and blood, and of biochemical changes in urine and blood and histological changes in the lungs. Anaemia developed, together with changes in the blood lipoids and serum proteins indicative of deranged nitrogen metabolism, and alteration of arterial oxygen tension. The only cellular changes were in the lungs. In many species the pulmonary response to exposure resembled closely that seen in human acute beryllium poisoning. Photographs are reproduced.

The authors conclude that inhalation of beryllium gives rise to a complex set of responses and that these are due to the action of beryllium itself. Toxic responses occur at exposure and tissue concentrations far below those usually encountered in chemical toxicity.

A. Thelwall Jones.

Acute Inhalation Toxicity of Beryllium. II. The Enhancing Effect of the Inhalation of Hydrogen Fluoride Vapor on Beryllium Sulfate Poisoning in Animals. STOKINGER, H. E., ASHENBURG, N. J., DEVOLDRE, J., SCOTT, J. K., and SMITH, F. A. (1950). *Arch. industr. Hyg. occup. Med.*, **1**, 398.

Potentially toxic dusts or vapours encountered in industry are frequently not single compounds but mixtures of various substances. An example occurs in the manufacture of beryllium, in which hydrogen fluoride and other fluorides are present. Experiments were made on rats to determine the enhancing effect of the inhalation of hydrogen fluoride vapour on beryllium poisoning. Experimental details are fully given. The authors conclude that inhalation of hydrogen fluoride vapour by rats at a concentration of 8 mg. per cubic metre enhanced twofold the toxicity of a soluble beryllium salt also inhaled by these rats. Inhalation of a beryllium salt as a mist at 9 mg. per cubic metre alternately with hydrogen fluoride increased the deposition of fluoride in tooth and bone to 1·3 times that from the inhalation of similar quantities of hydrogen fluoride vapour alone at 8 mg. per cubic metre. The mechanisms by which these effects occur are discussed.

A. Thelwall Jones.

Acute Toxicity of Inhaled Beryllium. III. Observations Correlating Toxicity with the Physicochemical Properties of Beryllium Oxide Dust. HALL, R. H., SCOTT, J. K., LASKIN, S., STROUD, C. A., and STOKINGER, H. E. (1950). *Arch. industr. Hyg. occup. Med.* **2**, 25.

An account is given of the effects on experimental animals of inhalation of air containing beryllium oxide dust in various grades and concentrations for periods of 6 hours, 5 days a week, the total exposure ranging from 56 to 360 hours. No pathological changes were found post mortem, save in the lungs. Tissue analysis showed by far the greatest amounts of beryllium in the lungs and pulmonary lymph nodes ; small quantities occurred in liver, spleen, and bone.

The effects observed both before and after death varied with the grade of beryllium oxide inhaled, particularly with the particle-size and state of aggregation. Dyspnoea, anorexia, and loss of weight were observed in all animals exposed to high concentrations of beryllium. Death was accompanied by cyanosis. In view of the fact that human chronic pulmonary berylliosis produces cor pulmonale, haematological studies were made ; polycythaemia was not noted, but a macrocytic hypoplastic or aplastic anaemia is described. Hyperglobulinaemia was noted, similar in degree to that in other chronic granulomatous diseases. Diffuse and focal pulmonary granulomatosis with fibrosis, as described in chronic berylliosis in human subjects, were not found, the animals showing acute inflammatory bronchial and pulmonary changes.

L. W. Hale.

The Influence of Beryllium and its Compounds on Man. FRANT, R. (1950). *Ned. Tijdschr. Geneesk.*, **94**, 1474.

The light metal beryllium is becoming important in industry in Holland. It is used in the fabrication of fluorescent tubes. Acute irritation of the skin and mucous membranes is known to be caused by beryllium-containing vapours. Removal of the patients from the area of exposure is sufficient for cure. Implantation of small beryllium particles in the skin causes the development of subcutaneous granulomata and ulcers, which require excision. The inhalation of beryllium-containing fumes produces acute interstitial pneumonitis with pyrexia, dyspnoea, cyanosis, and pain in the chest. Though the prognosis is favourable, occasionally death occurs through pulmonary oedema. Recovery takes 1 to 4 months. In a small percentage of cases the lung disease becomes chronic and may give rise to a dramatic

change in the patient's appearance. Certain " trigger " conditions may cause an acute exacerbation of the chronic pneumonitis. These cases are described as cases of delayed chemical pneumonitis. The chronic fibrosis of the lung finally causes insufficiency of the right ventricle, increasing cyanosis, clubbing of fingers, and enlargement of liver and spleen. The radiographic appearance of the lung varies from a ground-glass appearance to diffusely spread nodules. The histological picture resembles that in sarcoidosis. Treatment with BAL is ineffective. Renal calculi develop in 13% of cases. *F. Bloem* (*Excerpta Medica*).

Distribution and Fate of Cadmium in the Animal Body. POTTS, A. M., SIMON, F. P., TOBIAS, J. M., POSTEL, S., SWIFT, M. N., PATT, H. M., and GERARD R. W. (1950). *Arch. industr. Hyg. occup. Med.*, **2**, 175.

A series of experiments is described in which the isotope cadmium 115 (^{115}Cd) was administered to animals, which were killed at varying intervals so that the cadmium content of the organs could be estimated after ashing. When small doses (LD 100) of radioactive cadmium chloride were given to mice by inhalation for about half an hour, most of the cadmium retained by the animals had passed the pulmonary barrier at the termination of exposure. During the 12 hours after exposure the lung content of cadmium first fell to 65% of its original value and then remained constant more or less indefinitely. During the same period the cadmium content of the liver and the kidney rose to a constant value. Cadmium was highly concentrated in the lungs and liver after inhalation, but the kidney and liver showed the highest values after injection. Cadmium is normally excreted in the intestine, and the values for the gut and gut washings rose to a high peak about 2 hours after inhalation, and then gradually fell to zero.

If BAL is administered before cadmium chloride is inhaled, the lung retains about twice as much as without medication and mortality is increased. On the other hand, when BAL is given after exposure the lung content of cadmium is similar to that found when cadmium chloride is inhaled without any subsequent treatment, but there is a marked increase in the level in the urine. Mortality was reduced by up to 100% by this last measure.

It is suggested that the toxicity of inhaled cadmium depends on the amount of bivalent cadmium ion available for combination with lung constituents. Cadmium sulphide is virtually non-ionized, and it is non-toxic after inhalation. The cadmium–BAL complex is considerably more dissociable than cadmium sulphide, but it is less ionized than the soluble salts, such as the chloride. When BAL is given before cadmium chloride is inhaled, the formation of the cadmium–BAL complex traps large amounts of cadmium in the lungs, these being then slowly liberated over a long period causing severe pulmonary damage. If BAL is given after exposure the lung content of cadmium does not differ from that found when no medication is used, but the cadmium present in the lung combines with BAL and ionization is suppressed, with consequent reduction of pulmonary damage.

The cadmium–BAL complex is apparently excreted by the kidneys, and this accounts for the large amount of cadmium in the urine when BAL is given after exposure.
W. K. S. Moore.

The Toxicity of Sulfur Pentafluoride. GREENBERG, L. A. and LESTER, D. (1950). *Arch. industr. Hyg. occup. Med.*, **2**, 350.

Sulphur pentafluoride (SF_5) was found to cause intense irritation in the lungs of rats. The substance itself is not an irritant and so it was assumed that the toxic effects were due to products of hydrolysis, such as hydrogen fluoride. The action is therefore comparable in type to that of phosgene, but is very much more intense—whereas 1 part per million is given as the maximum permissible concentration of phosgene for prolonged exposure, sulphur pentafluoride is lethal at this concentration. It is concluded that the maximum permissible concentration for sulphur pentafluoride should be 0·01 part per million. *W. K. S. Moore.*

Toxicity of the Tetrachlorodifluoroethanes. GREENBERG, L. A. and LESTER, D. (1950). *Arch. industr. Hyg. occup. Med.*, **2**, 345.

Neither of the tetrachlorodifluoroethanes show toxic properties when ingested by rats. The vapours of both compounds caused anaesthesia at a concentration of 1·5% by volume, and were rapidly fatal at 2%. The symmetric compound 1 : 1 : 2 : 2-tetrachloro-1 : 2-difluoroethane is a pulmonary irritant at concentrations of 1·0% by volume for short exposures, while at 0·5% it causes death after 18 hours' exposure. It is suggested that the maximum permissible concentration for both compounds should be 0·1% by volume of the vapour.
W. K. S. Moore.

The Toxicity of Sulfur Hexafluoride. LESTER, D. and GREENBERG, L. A. (1950). *Arch. industr. Hyg. occup. Med.*, **2**, 348.

Sulphur hexafluoride (SF_6) is a colourless and odourless gas which is used as a dielectric in high-voltage equipment. Rats exposed to an atmosphere containing 80% of the gas by volume showed no ill-effects and it appears to be physiologically inert. *W. K. S. Moore.*

Acute and Chronic Toxicity of Some Halogenated Derivatives of Methane and Ethane. LESTER, D. and GREENBERG, L. A. (1950). *Arch. industr. Hyg. occup. Med.*, **2**, 335.

Fluorine-chlorine derivatives of methane and ethane have recently come into commercial use as refrigerants and aerosol propellents, and have even been considered as possible anaesthetics. An investigation of the acute and chronic toxic action of some of these derivatives in rats is described, the substances investigated being 1 : 1-difluoroethylene, fluoroethylene, dichlorodifluoromethane, 1 : 1-difluoroethane, 1-chloro-1 : 1-difluoroethane, trichlorofluoromethane, and 1 : 1-difluoro-1 : 2-dibromoethane. The boiling points and toxic action of these chemicals increase in the sequence in which they

are given above, and the author suggests an explanation for this correlation. He assumes that similar blood concentrations of any of these compounds will produce the same effect. Now as the boiling point increases and, presumably, the vapour pressure decreases, so also will the blood–air partition ratio increase. Thus the smaller the vapour pressure becomes the greater will be the blood concentration.

In most cases toxic symptoms occurred at a dose closely approximating to the anaesthetic level, while the ethylene derivatives did not produce anaesthesia even at a concentration of 80% by volume, so that these substances would clearly be unsuitable for use as volatile anaesthetics. 1 : 1-difluoroethane, 1-chloro-1 : 1-difluoroethane and 1 : 1-difluoro-1 : 2-dibromoethane caused pulmonary congestion and oedema in addition to anaesthesia, but none of the compounds examined caused hepatic damage. A maximum allowable concentration is suggested for each compound. *W. K. S. Moore.*

Thiocyanate Effect following Industrial Cyanide Exposure. Report of Two Cases. HARDY, H. L., JEFFRIES, W. MCK., WASSERMAN, M. M., and WADDELL, W. R. (1950). *New Engl. J. Med.*, **242**, 968.

The authors describe in detail 2 cases of changes in the thyroid in persons exposed to cyanide fumes from the case-hardening process. The highly toxic cyanide ion is rapidly converted in the body to the relatively non-toxic thiocyanate ion, which is excreted irregularly by the kidneys. Persons constantly exposed to cyanide therefore accumulate thiocyanate in the tissues, and it is reasonable to suppose that, by interfering with the iodine metabolism, this may lead to the development of goitre. There is, in fact, clinical evidence that the treatment of hypertensive patients with thiocyanate may give rise to goitre. It is suggested, therefore, that in the 2 cases reported the goitre was due to thiocyanate accumulation, and further that various symptoms previously attributed to the direct effects of chronic cyanide poisoning may, in fact, be due to this cause, a theory which is well supported by clinical and experimental observations. The authors' purpose in publishing this article is to encourage clinical interest in detoxification mechanisms in the body. *A. Thelwall Jones.*

Industrial Poisoning with Nitrochlorobenzol. SPAUN, J. (1950). *Ugeskr. Læg.*, **112**, 1046.

This paper describes the clinical and laboratory findings in 7 young males suffering from chronic industrial poisoning with nitrochlorobenzol. Six were admitted acutely ill, 2 of them direct from their place of work, while in 2 others the onset followed the intake of alcohol. Malaise and cyanosis were present in all cases ; other commonly described symptoms and signs, such as headache, giddiness, and pallor, were less constant. The temperature was raised in 3 cases and subnormal in one. Six were mildly anaemic, with haemoglobin values between 81 and 88% (Zeiss) ; in the seventh the anaemia was more severe (66%). Treatment consisted in the administration of stimulants and oxygen. Most patients could be discharged within one week ; five were followed up 6 months later and showed no evidence of after-effects. *W. G. Harding.*

Ulceration of Larynx and Vocal Cords in the Manufacture of Chromium Derivatives. MANCIOLI, G. (1950). *Rass. Med. industr.*, **19**, 170.

The author studied a group of 107 men, working at a factory in which sodium bichromate was made, whose ages ranged from 17 to 51 years. They had worked for from 1 month to 20 months in the factory.

Subjective symptoms ranged from local pruritus and burning to sneezing, epistaxis, and rhinorrhoea, the nasal secretion being yellow in colour. Pain was felt in the nose or frontal region ; hoarseness, sore throat, and cough were common. Inflammatory changes were seen in the nasal mucosa, and septal perforation was common ; a chronic pharyngitis was often seen with swelling of the cords, ulceration being common on these and on the epiglottis also. The area below the cords was sometimes examined and the upper trachea was seen to be red and oedematous.

The working sites were often covered by a yellowish dust, and the air was dusty and sometimes full of fumes. Although the works were not crowded, the methods of exhaust ventilation were inadequate to prevent gross contamination of the personnel. *G. C. Pether.*

Pharmacologic Tools in the Study of the Mechanism of Uranium Poisoning. HODGE, H. C. (1950). *Arch. industr. Hyg. occup. Med.*, **2**, 300.

In this paper the author reviews in summary the various methods of investigation which are being employed in the study of uranium poisoning. A brief description of each method is given, with relevant references. Fluorometric analysis, ultrafiltration, protein precipitation, bone adsorption studies with radioisotopes, radioautography of the kidney, renal clearance studies, estimation of enzyme activity in urine, and the study of cell physiology and histopathology are the types of investigation cited.

As a result of these studies (mainly in rats) it has been found that when the uranyl ion enters the body it is transported in the blood partly as a non-diffusible protein complex and partly as diffusible bicarbonate complex. A substantial part of the uranium is deposited in bone, displacing calcium ions, but most of it is excreted by the kidney. The kidney tubule is damaged in the distal half of its proximal segment, and if the animal survives regeneration of the tubule cells takes place. Death occurs as a result of uraemia. Fortunately there is little absorption of uranium by way of the skin or gastro-intestinal tract. *W. K. S. Moore.*

Results of Physical Measurements in a Case of Occupational Radium Poisoning with a Fatal Outcome. MUTH, H., and ROTH, H. H. (1949). *Strahlentherapie*, **80**, 271.

While working with radium and mesothorium, a chemist acquired radium poisoning which ended fatally. In the years 1932–37 he was examined at intervals ; results of all investigations including blood count were

normal. In the autumn of 1940 the first clinical changes were observed, rales appearing over the apices of the lungs, but other findings were normal. In the beginning of 1943 a rapid change for the worse was noted; the haemoglobin content was 17%, erythrocyte count 800,000 per c.mm., and leucocyte count 1,800. Anti-anaemic therapy with numerous transfusions did not stop the progress of the disease. Radiographs of the lung showed in the upper field striae and patches of a bronchopneumonic character. Death occurred in 1944. Necropsy revealed aplastic anaemia, petechial haemorrhages in the pleurae, pericardium and endocardium, and marked pulmonary fibrosis, with pale bone marrow. The radioactivity of different parts of the body was determined with sensitive counters, and the whole content of radioactive substance was estimated at about 3·5 μg., the bone marrow being about 400 times more radioactive than the heart muscle. The patient's clothes showed marked radioactivity. The combination of aplastic anaemia and pulmonary fibrosis is unusual. Since 1937 irradiation ulcers had also been present in the skin of the hands. *Y. T. Seuderling* (*Excerpta Medica*).

Acute Poisoning inside Oil Tanks and its Prevention. WALCZYNSKI, J. Z. (1950). *Med. Pracy*, **1**, 60.

The cleansing of oil tanks is dangerous because of the possibility of poisoning; the latter can be avoided by strict application of regulations. Despite the removal of fumes from tanks before the process of cleaning, it should be borne in mind that some vaporized oil derivatives may be left behind.

The author describes 14 cases of acute poisoning, which took place during the cleaning of oil tanks and other procedures; the poisoning was due to derivatives of oil. The paper contains details of the pathological findings as well as the clinical symptoms in cases of acute poisoning by oil derivatives. Special attention is paid to the toxicology of benzine poisoning and to safety measures. *J. W. Czekalowski.*

Arsine Poisoning in the Smelting and Refining Industry. MORSE, K. M., and SETTERLIND, A. N. (1950). *Arch. industr. Hyg. occup. Med.*, **2**, 148.

In January, 1949, 4 deaths from arsine poisoning occurred in Indiana, as a result of handling arsenic-bearing dross. The dross had been supplied by a firm in Illinois, and this led to an investigation of the smelting and refining plants in the latter State. During the progress of this investigation, 2 deaths occurred in an Illinois factory. The 2 men who died were engaged in ladling the dross formed in a large vessel of molten lead, to which aluminium had been added to remove antimony and arsenic which were present as impurities. The molten dross was placed in ingot moulds, which were then sprayed with water to facilitate cooling. Both men became ill 2 days after this work, and severe haemolytic anaemia and anuria developed. One man died after 7 days and the other after 3 weeks.

Detailed investigation into the chemical reactions of this dross, which consisted of lead (57·96%), aluminium (3·58%), antimony (10·95%), and arsenic (0·91%) revealed that the addition of water resulted in the evolution of hydrogen and arsine. High concentrations of arsine (300 p.p.m.) were found in the neighbourhood of the ingot moulds containing the dross, if there was any moisture present.

Addition of water to mixtures of finely atomized lead and aluminium caused evolution of hydrogen and some oxygen. When arsenic was added to the mixture arsine was produced as well as hydrogen. The suggestion is made that the difference in potential between the individual lead and aluminium particles created the conditions necessary for electrolysis, and the nascent hydrogen produced then reacted with the arsenic present to form arsine.

The form of the dross could be modified by adding sawdust as well as aluminium to the molten lead during purification; when the resulting "sawdust concentrate dross" was roasted in an oven for an hour at 1800°F. (982°C.) it became "stabilized" and would no longer produce arsine on contact with water.

W. K. S. Moore.

Arsine Poisoning. A Study of Thirteen Cases. PINTO, S. S., PETRONELLA, S. J., JOHNS, D. R., and ARNOLD, M. F. (1950). *Arch. industr. Hyg. occup. Med.*, **1**, 437.

Thirteen cases of arsine poisoning are described, and further details are given by colleagues of the authors in another paper in the same issue.

No reliable estimate of the arsine concentration of the air was possible, but the fumes were generated by moisture reacting with aluminium arsenide in metal dross. The usual symptoms of arsine poisoning were seen and these included abdominal cramps, nausea and vomiting, and the passage of a dark-red urine. The supernatant fluid from centrifuged blood samples was found to be dark red. The cases varied in severity and the first four patients to enter the hospital died.

The laboratory findings are given in detail and many interesting factors are recorded. In the microscopical examination of urine no haemoglobin casts or cylindroids were found. There was no apparent correlation between the arsenic content of the urine and the amount of haemoglobin in the urine. Thus the urinary arsenic content is no measure of the degree of blood destruction. Initial leucocyte counts were made in 7 cases before any treatment was given and these revealed a leucocytosis ranging from 13,000 to 29,000 per c.mm. The count returned to normal in all cases, but a further rise occurred on the fourth to sixth day after poisoning. Haematocrit readings showed evidence of rapid destruction of erythrocytes, haemoglobin accumulating in the plasma much faster than it could be removed or excreted. There was a rise in the non-protein nitrogen content of the blood, indicating that haemoglobinuria damages the excretory power of the kidneys. In all cases from the beginning serial electrocardiograms were taken, and changes were noted, particularly in the T waves.

Only in one case was there a suggestion of jaundice. Treatment was mainly by transfusion of whole blood and there was no indication that such added blood was haemolysed. In the opinion of the authors, British anti-

lewisite (BAL) had no influence on the course of the erythrocyte destruction produced by arsine. Whether BAL aids in preventing other disorders, such as peripheral nerve involvement, is as yet uncertain. Details are given of the necropsy evidence in 4 cases, death being due to sudden myocardial failure. The distribution of arsenic in the tissues is recorded, the figures agreeing in general with those in previous investigations.

A. Thelwall Jones.

Cutaneous Effects of Accidental Ingestion of Arsenic. BUREAU, Y., RINCKENBACH, —., and BARRIÈRE, H. (1950). *Ann. Derm. Syph., Paris*, **10**, 385.

An account is given of 21 cases of chronic arsenical poisoning out of a total of 80 resulting from drinking contaminated wine. The cutaneous effects seen in these cases are summarized as follows: (1) Erythemata: (*a*) Localized; exposed parts, sometimes pellagroid; extremities, acrodynic type; intertrigo. (*b*) Generalized; scarlatiniform. (2) Vesicles and bullae: eczematous; herpetiform; zosteriform. (3) Keratoses: follicular; keratoderma of palms and soles; leukokeratotic appearance of mucosae. (4) Dyschromia: localized or generalized melanoderma. (5) Lesions of mucous membranes: enanthemata; eosions; ulceration.

[No reference is made to the interesting study by Zinny and Vivaldo "Arsenicismo Cronico Regional Endemico Producido por las Aguas de Consumo" (Lopez, Buenos Aires, 1942).] *James Marshall.*

Psychical Disturbances in Chronic Mercury Poisoning. GOUSENBERG, M. (1950). *Mschr. Psychiat. Neurol.*, **120**, 38.

It is pointed out that the effects of mercury on the young, as observed by Feer in those susceptible to calomel, do not resemble those noted in adults. In children there may be obvious allergy to the metal which lessens or disappears in later life. Some errors have arisen in industrial poisoning because the effects of other toxic substances, also present in a factory, have been overlooked. Among such are nitrous fumes, often found in explosives factories. A useful measure of the possibility of mercury poisoning may be gained by estimating the loss of metallic mercury in a given process; in some works several tons are not accounted for each year, so that the employees are clearly exposed to considerable hazard. Stock noted the amount of mercury excreted by 9 workers; the urinary excretion varied from 600 to 16 μg. per litre. The amount recovered in water used for washing the hands is also a useful index of the degree of exposure. In many employees the excretion greatly exceeds normal figures yet there are no symptoms of poisoning, suggesting some acquired tolerance to the metal.

The psychic changes noted in mercurialism have been briefly described as an anxiety state with a tendency to panic. Hurried and uncertain movements betray a feeling of insecurity. Blushing and pallor are noted, with an expression of confusion. There is lability of mood with flight of ideas, and tremor increases when the subject thinks that he is observed. Movements needing some dexterity are difficult. Lassitude, insomnia, nightmare, and hallucinations may occur. In addition to symptoms presumably of organic origin apathy and discouragement are not uncommon. Fits of temper, immediately regretted, may develop, but there may be timidity and hesitation. Loss of libido may have serious marital consequences. Rorschach's test has been used to demonstrate affective changes. Intelligence is somewhat diminished, memory weakened, and attention poor.

The author examined 11 patients, all of whom were making insurance claims. It appeared probable that many of the symptoms described were hysterical or due to a desire to substantiate a claim for compensation. Some of the patients complained of fatigue and exhaustion, yet were able to perform fairly heavy work or to move briskly after leaving the clinic. Others complained of headache and the early onset of fatigue after intellectual effort. Loss of concentration, depression, and irritability were frequent.

Preventive measures may be difficult to apply. An experienced workman may resent and try to avoid transfer to a task in which exposure to mercury is less or absent. The factory doctor should watch for mercurial tremor, nervousness, intolerance of alcohol, or loss of libido. A handwriting test performed after muscular effort is a valuable guide to the degree of incoordination present. After recovery from an attack of mercurialism a subject is more sensitive to the metal than before. Delayed poisoning may also occur. Many employees, clearly affected by the poison, may cheerfully continue at work with insight into their condition, knowing that they are receiving higher pay because of the danger involved. Those of poor intelligence but aware of their disabilities may exaggerate their symptoms and develop a persecution state. In reviewing the literature the author was struck by the great diversity of signs and symptoms; no clear-cut syndrome can really be described.

It is necessary in all cases to take into account the individual peculiarities of the patients, notably in the psychiatric sense, for this will determine to a great degree the symptoms developing in mercurialism.

G. C. Pether.

The Toxicity of Vapours of Organic Mercury Compounds (Ethyl Mercuric Phosphate and Ethyl Mercuric Chloride) under Acute and Chronic Conditions. TRAKHTENBERG, I. M. (1950). *Gigiena*, No. 6, 13.

As a preservative of grain various chemical substances have been employed. Among these are the ethyl phosphate and ethyl chloride compounds of mercury. Experiments were performed on white mice to determine the toxicity of these substances. Analyses were also made on collective farms and near or in grain stores. Some of these were performed in ventilated places or in the open, others under cover. It was found that mice died quickly if exposed to a concentration of 0·01 to 0·04 mg. per litre of air in the case of the ethyl mercury phosphate and 0·03 to 0·04 mg. per litre in the case of ethyl mercury chloricde.

Both acute and chronic mercury poisoning occurred, depending on the concentration of the fumes and the

period of exposure. After 2 weeks or so the animals' behaviour changed; they became drowsy and refused food, and paralyses and tremor or clonic convulsions appeared later.

It is concluded that in work with grain even a trace of mercury should be avoided, because a concentration as low as 0·0001 mg. per litre is dangerous.

G. C. Pether.

INDUSTRIAL PHYSIOLOGY

Coproporphyrinuria as an Index of Lead Absorption. JOHNSON, W. S. and WHITMAN, N. E. (1950). *Arch. industr. Hyg. occup. Med.*, **2**, 170.

Lead analysis and an arbitrary assessment of the amount of coproporphyrin III in the urine were carried out in 294 cases of suspected lead poisoning. Coproporphyrin was determined by acidifying 5 ml. of urine with 6 drops of glacial acetic acid, shaking the urine with 5 ml. of ether, and examining the ether layer for fluorescence under ultraviolet light (3,660 A) in a dark room. The degrees of fluorescence were compared with those obtained from standard samples of coproporphyrin in urine, and the results were divided into six ascending grades. The comparison between the lead values found by analysis and the fluorescence grades is shown in a table and a scattergram.

The upper and mean values for lead content of the urine increased with the grade of fluorescence. It is suggested that with the first grade of fluorescence (0·5 mg. of coproporphyrin per litre) the urine contains no more than 0·18 mg. of lead per litre except in rare instances. Increased coproporphyrin excretion was found to follow lead exposure by 12 to 18 days, whereas elevation of the lead content of urine may occur in only 5 days. [The scattergram suggests that only the 6th grade of fluorescence provides definite evidence of increased lead excretion.] *W. K. S. Moore.*

Mercury and Lead Storage in Human Tissues. With Special Reference to Thromboytopenic Purpura. BUTT, E. M. and SIMONSEN, D. G. (1950). *Amer. J. clin. Path.*, **20**, 716.

The mercury content of liver and kidney tissue obtained at necropsy in 134 cases was estimated by means of a dithizone method devised by one of the authors and said to give results which are accurate to within 10 μg. of mercury per 100 g. of wet tissue. The cases are divided arbitrarily into 6 groups, according to the cause of death and history of therapeutic or other intake of mercury: I. Miscellaneous. No known intake of mercury. II. Cases in which death may have been related to poisoning with a heavy metal (Pb, As, Cu, or Sb), including one case of lead poisoning with terminal acute mercurial nephritis due to application of a mercurial ointment to a skin ulcer. III. Cases of renal failure from various causes, with or without history of contact with mercury. IV. Cases of congestive heart failure treated with mercurial diuretics. V. Cases of other conditions treated with mercurial diuretics. VI. Cases of acute thrombocytopenic purpura, developing in 3 out of 13 cases during treatment with a mercurial diuretic and with a history of contact with mercury in one further case. The mean of the results and the ratio of the mercury content of the liver to that of the kidney in each of these groups are set out as follows:

Group	No. of Cases	Mercury Content in mg. per 100 g.		Liver/Kidney Ratio
		Liver	Kidney	
I	69	0·006	0·075	1 : 12
II	11	0·058	0·192	1 : 3·3
III	8	0·105	0·425	1 : 4·1
IV	22	0·757	8·619	1 : 11·3
V	11	0·659	3·171	1 : 4·8
VI	13	0·287	0·704	1 : 2·4

In the 13 cases of fatal acute thrombocytopenic purpura the lead content of the liver and kidney was 0·127 mg. and 0·227 mg. per 100 g. respectively. The authors comment on the unusually high levels of mercury found in the cases of thrombocytopenic purpura not associated with treatment with mercurial diuretics. They infer that mercury may have a poisonous effect on germ cells. *A. Michael Davies.*

Influence of Lead Absorption on the Ratio of Large to Small Lymphoid Cells. SHIELS, D. O. (1950). *Med. J. Aust.*, **2**, 205.

The effects of lead on cells of the lymphocyte series are described. The study was carried out on the employees of a large new storage-battery works, and also on men engaged in the mining, milling, smelting, and assaying of lead ore. No worker was exposed to tetraethyl lead. The ratio of large lymphocytes (over 10 μ in diameter) and monocytes to small lymphocytes was determined. It was shown that among new workers entering the industry the absorption of lead, as determined by increasing concentration of lead in the urine, caused an increase in this ratio of monocytes and large lymphocytes to small lymphocytes. This increase, however, does not go on indefinitely; too much lead in the system acting for too long causes a fall in the ratio to within the range of the ratio of non-exposed persons. If, therefore, the ratio falls to less than 2·0 lead poisoning is imminent, if not already present.

Recovery from lead poisoning is accompanied by a rise in the ratio above 2·0 as the lead is excreted in the urine; as further lead is excreted and the concentration reverts to that in the non-exposed person, the ratio again falls to the range of that in the non-exposed person. The author considers that this cell ratio is a valuable guide in the prevention and diagnosis of lead poisoning.

K. M. A. Perry.

Biochemical Changes in Acute Beryllium Poisoning. ALDRIDGE, W. N., BARNES, J. M., and DENZ, F. A. (1950). *Brit. J. exp. Path.*, **31**, 473.

The biochemical effects of intravenous administration of beryllium (0·5 to 0·75 mg. per kg.) to rats and rabbits have been studied. The animals died within 4 days, death being preceded by low blood sugar levels which

were the result of extensive liver necrosis. Renal damage was insignificant. The rate of removal of glucose and lactic acid from the blood was slightly reduced 24 hours after the beryllium was given, as shown by tolerance curves. During the following 48 hours the ability to excrete beryllium became greatly impaired. Focal necrotic lesions in the liver were apparent after 24 hours and there was a significant fall in the liver potassium level. Serum and liver alkaline phosphatase levels were raised during the later phases and liver arginase level fell. *F. W. Chattaway.*

Absorption and Elimination of Inhaled Benzene in Man. SRBOVÁ, J., TEISINGER, J., and SKRAMOVSKY, S. (1950). *Arch. industr. Hyg. occup. Med.*, **2**, 1.

An account is given of 27 experiments carried out at Charles University, Prague, on 23 volunteers, who inhaled mixtures of benzene vapour and air, the concentration of benzene ranging from 150 to 350 μg. per litre. After periods of inhalation of 2 to 3 hours, the benzene content of the subject's blood, urine, and exhaled air was estimated polarographically until the values were no longer significant. It was found that different subjects retained amounts of benzene varying between 33 mg. and 198 mg. during 2 hours' inhalation ; the reason for variation in absorptive capcaity is discussed. No subjective symptoms resulted. A considerable part of the absorbed benzene was excreted by the lungs (16·5% in 6½ hours), and a much smaller quantity by the kidneys (0·05% in 12 hours), the remainder being retained and metabolized. Equations are given by which the time necessary for inhaled benzene to be eliminated from the body may be calculated.
L. W. Hale.

A Study of the Excitability of Respiratory and Sympathetic Pulmonary-vascular Centres in Silicosis. SANTENOISE, D., MATTEI, J., PERRET, A., and ROCHE, M. (1950). *Arch. Mal. prof.*, **11**, 277.

The excitability of the respiratory centre was judged by requiring subjects to breathe air containing increasing proportions of carbon dioxide ; respiratory movements were measured mechanically, and the concentration of CO_2 was noted at which a considerable increase in amplitude of respiration occurred (an earlier and slighter increase in some subjects was discounted, as probably resulting from direct irritation of pulmonary sensory nerve endings). In normal subjects the depth of respiration increased when the CO_2 content of the inspired air was 2 to 4%. Of 55 untreated miners with silicosis, 54 showed no response to 4% CO_2, while 8 required over 8% for increase in respiration. A similar group of miners who had been treated in hospital for several months showed reactions at levels closer to the normal.

Compression of the abdomen led to rise in systemic diastolic pressure and diminution in systolic pressure in 108 out of 127 silicotic miners who had received no treatment. This type of reaction is interpreted by the authors as evidence of hyperexcitability of sympathetic vasoconstrictor nerves in the pulmonary circulation. A similar type of reaction was observed in 21 of 36 miners who appeared to have early silicosis and were still able to work.

The authors suggest that these modifications of nervous excitability may precede the radiological appearance of silicosis, and may afford the earliest means of diagnosis. On the other hand, it is possible that subjects who possess an abnormal excitability may be more prone to develop silicosis. *H. E. Harding.*

INDUSTRIAL LUNG DISEASES

The Effect of Aluminium Dust on the Animal Organism. IVANOVA, M. G. and OSTROVSKAJA, I. S. (1950). *Gigiena* No. 4, 21.

The authors consider that insufficient study has been devoted to the value of aluminium inhalations in preventing silicosis. The experiments performed in Canada by Denny, Robson, and Irvin showed that quartz dust was absorbed more slowly from lung tissue if aluminium had previously been inhaled, but in the authors' view the period of observation was too short to permit of adequate conclusions ; they suggest that commercial views have prevailed against scientific evidence.

They also think that a fundamental weakness in this method of prevention is the impossibility of gauging the amount of aluminium dust which should be used. Even if, theoretically, the addition of 1% of aluminium dust to the silica prevents or lessens absorption, there is no absolute proof that this is the ideal proportion. They do not agree that the introduction of aluminium dust into the body is essentially harmless, as some Canadian authors have suggested. According to Gardner aluminium has no retarding effect on silicosis, for the disease progresses despite its use. Lanza and Berry have noted that the introduction of aluminium into the blood stream or under the skin may have a toxic effect, paralyses developing in the central nervous system and the heart. In mammals tremor, loss of sensation, and progressive paralysis have been observed. Aluminium preparations are used as sclerosing agents, and this action is due to the formation of dense tissue round the particles and to changes in the tissue protein in contact with aluminium solutions.

Lung lesions are not uncommon in workers in aluminium factories, a matter worth consideration when the therapeutic use of the metal is contemplated.

The authors experimented on white rats and rabbits by : (1) intratracheal administration of aluminium powder in physiological saline (white rats only) ; (2) intravenous injection of the same solution (rabbits) ; (3) exposure to the dust in closed chambers (rats and rabbits). A very fine dust was used with particle size not exceeding 2μ. For the intratracheal injections 0·01 g of powder was suspended in 3 ml. of saline, of which suspension one-third was used.

The first experiments showed that the powder was far from harmless. In a rat killed 3 months after the intratracheal injection, nodules of fibrous tissue were found, mostly in the terminal bronchioles. Some of this tissue showed hyaline change with obliteration of vessels. Attempts were made to determine the first

pathological changes and the sequence of changes. In particular the fate of the dust particles, and the possible phagocytosis of these, were of interest. It was hoped to discover why fibrotic changes occurred, and also the general systemic effect of any aluminium which might be absorbed into the circulation.

The second series of rats was exposed to aluminium dust averaging 5,000 particles per ml. for half an hour every day. Some were allowed to die naturally and others were killed at intervals. Others were exposed to dust for 8 hours daily for 15 days. At necropsy many cells in the alveoli and adjacent tissues were filled with dust, so that the nuclei were often indistinguishable. There was marked local leucocytosis with obliteration of some of the finer air spaces. Rats killed after 2 or 3 months showed, in addition to the changes described, marked peribronchial fibrosis with perivascular thickening and narrowing of the lumen.

In all animals studied the dust was evidently taken up by the alveolar epithelium and carried into the adjacent tissues, but the regional lymph nodes were almost free of dust because of blocking of the intercommunicating lymphatics. Sclerosis continues even when exposure to dust has ceased. Similar results were noted in rabbits. It is evident that the changes represent a severe aluminosis with, in some cases, superadded pneumonic changes and death. Pathological changes were seen in the kidneys and sometimes in the heart and other organs. It is concluded that the prophylactic use of aluminium needs further study, as also the lungs of the workers handling this metal. *G. C. Pether.*

The Effect of Wood Dust on the Lungs. VALLANDER, A. (1950). *Nord. Med.*, **44**, 1315.

The author has examined the workers employed for 10 years or more in the Varnamo timber industry (81 in all). Apart from radiological evidence of calcified primary foci and old pleurisy (6 cases) only 2 men were found to have abnormal signs in the chest: one had asthma, the other chronic bronchitis with emphysema and pulmonary fibrosis. The author considers that exposure to sawdust is unlikely to have been an aetiological factor in either of these cases and that there is no risk of pneumoconiotic changes associated with such employment. *W. G. Harding.*

Pneumonokoniosis in Queensland Foundries. GORDON, D. (1950). *Med. J. Aust.*, **2**, 217.

In 1947 and 1948 seventy-six Queensland foundries, employing 1,100 men, were studied from the point of view of pneumoconiosis. They included iron and non-ferrous foundries and one steel foundry. Moulders who had worked more than 14 years and fettlers who had worked more than 4 years were considered suitable for examination. Out of 388 such men, 359 agreed to cooperate; 203 moulders, 67 fettlers, 42 moulders' assistants, and 47 other foundry workers were examined. Thirteen of these workers were considered to have silicosis, largely on radiological grounds, and in addition 8 cases were considered doubtful. Of the 13 men, 4 were fettlers (2 steel), 4 were furnacemen (2 steel), 3 were moulders, one had a mixed occupational history, and one had been exposed to silica in the coal mines. The author suggests that these results confirm the well-known fact that in steel foundries the risk is much greater than in other foundries.

The great majority of cases were in elderly subjects. The men were fettlers or furnacemen and came from large foundries. Environment studies were carried out on the atmospheric dust; figures are given for dust counts from furnace chipping, the count ranging from 239 to 368 particles of dangerous size (of which 45% was free silica) per ml. of air. In the steel foundries highly siliceous parting powders were used.

The author contends that it is incorrect to regard a man with early silicosis as an invalid. *K. M. A. Perry.*

Pulmonary Manifestations of Gasoline Intoxication. A Review with Report of a Case. ZUCKER, R., KILBOURNE, E. D., and EVANS, J. B. (1950). *Arch. industr. Hyg. occup. Med.*, **2**, 17.

Commercial gasoline [benzine, petroleum spirit] is a mixture of volatile hydrocarbons and may cause irritative lesions of the skin, conjunctiva, or pharynx. It may be absorbed via the pulmonary or gastro-intestinal system, and may cause disturbances of the central nervous system. Pneumonia, bloody pleural effusion, emphysema, and lung abscess have been described as occurring after aspiration of gasoline. The literature is briefly reviewed and the case is described of a man who inhaled gasoline when attempting to siphon it from a car tank. He developed bilateral pneumonia, predominantly right-sided, with right pleural effusion, the onset being (as is usual) within 24 hours of inhalation. Recovery followed a period of critical pneumonic illness. *L. W. Hale.*

Lung Carcinoma in Iceland. DUNGAL, N. (1950). *Lancet*, **2**, 245.

Lung cancer is relatively rare in Iceland and was found in only 12 out of 337 necropsies on cases of carcinoma performed in Reykjavik during the period 1932–48. These 12 cases were distributed more or less evenly over the period concerned, affording no evidence that the disease is increasing in frequency in Iceland. If it be assumed that cigarette smoking is an important cause of lung cancer, this may explain the continued rarity of the disease in Iceland, where until recent years the consumption of cigarettes was relatively small. *R. A. Willis.*

INDUSTRIAL SKIN DISEASES

Skin Ulcers in a Factory in which Chromium Derivatives were Used. MENEGHINI, C. L. (1950). *Rass. Med. industr.*, **19**, 161.

It has been suggested that chromate ulceration only occurs when the acid or acid salts are [illegible] and that alkaline chromates do not cause it. The au[illegible] observed 120 workmen in a factory in which [illegible]ate was

used ; over 90% had skin lesions. These men had been working for from one month to 18 months, and those most affected were working in the lisciviation or filter departments. A high proportion of the lesions were on the hands, forearms, or feet. The ulcers were rounded or oval and the associated dermatitis, if present, was of the type often described. Sections showed that necrosis was so rapid that little repair had occurred. The author doubts whether any previously existing skin lesion is essential to the development of a chrome ulcer.

He performed numerous experiments which showed that the solutions of sodium chromate, sodium aluminate, and ferrous oxide which are mixed together in the first stages of manufacture have a *p*H of about 10 to 10·2. Application of this solution causes ulcers to form on the skin in 6 to 24 hours, and these become very obvious in 48 to 72 hours. The fluid obtained after centrifuging the strong saline solution mentioned is of a yellowish colour with a *p*H about 10, and does not readily cause skin lesions when applied. The solution of sodium bichromate, obtained by acidifying chromate solutions with sulphuric acid, has a *p*H of about 2·8. This solution causes ulceration almost indistinguishable from that caused by the alkaline solutions of chromates. Solutions of chromate and bichromate of equal strength, when applied to the skin, cause typical ulcers in the first case but only mild erythema in the second.

It may be said that alkaline chromates are very harmful, the acid solutions of bichromate are less so, and the dilute and slightly acid bichromate salts cause little or no damage. It appears that some views advanced by earlier authors can no longer be maintained.

Prophylactic measures should include the enclosure of all dangerous processes, the free use of masks, gloves, and protective boots, and the application of weak sulphurous acid to all exposed areas to reduce the chromate to an inactive oxide. Reducing agents should also be used freely in the works to neutralize any noxious material on machines or walls. A water-insoluble barrier cream has proved useful. *G. C. Pether.*

The Dermatophytoses of Great Britain. Report of a Three Years' Survey. WALKER, J. (1950). *Brit. J. Derm. Syph.*, **62**, 239.

A survey on as comprehensive a scale as possible of the present state of ringworm infections in Great Britain is described in detail. The types obtained from the various lesions, the numbers of each dermatophyte cultured, the age and sex incidence, and the probable sources of infection were investigated. The observations bear out the historical record of a great preponderance of the small-spore type of infection in England. In addition to an increase in indigenous infections, the return of large numbers of people from war-time service abroad has resulted in the introduction of new infections which themselves tend to become established and indigenous.

The author comes to the conclusion that the unavoidable relaxation of control and treatment of tinea capitis during the war has brought about a reversion to the conditions existing at the beginning of the century before the introduction of *x* rays. The geographical distribution of the different species of *Microsporum* is illustrated on a map. *G. B. Mitchell-Heggs.*

Treatment of Staphylococcal Skin Lesions with "Antifagin". GOLSHMID, K. L., and DATSKOVSKIJ, B. M. (1950). *Vestn. Vener. Derm.*, No. 4, 22.

"Antifagin" treatment was tried on 3,777 patients with suppurative skin lesions of up to 6 years' duration ; 2,946 patients were miners and 673 were metal workers. An average of 5 daily injections was given, together with local treatment. Complete healing after one course occurred in 1,789 out of 2,320 miners with furunculosis (77·1%) ; two courses produced a cure in 347, but were ineffective in 184 cases ; the total success rate was 92%. Underground workers did not respond so well to treatment, presumably because the disease was aggravated by the conditions of work. Cure was also obtained in 87% of 382 miners with carbuncle and 74·1% of 244 with hidradenitis, but treatment was ineffective in 150 cases of eczema with suppurative complications. Injection of antifagin was followed by a skin reaction in 70% of cases and a general reaction in 40%. Favourable effects were also obtained in the treatment of metal workers ; 92·7% of 594 patients with furunculosis were cured by one course of treatment. *D. J. Bauer.*

ENVIRONMENT

The Survival of Bacteria in Dust. I. The Distribution of Bacteria in Floor Dust. II. The Effect of Atmospheric Humidity on the Survival of Bacteria in Dust. III. The Effect of Light on the Survival of Bacteria in Dust. IV. Atmospheric Humidity and the Bactericidal Action of Ultra-violet Irradiation. LIDWELL, O. M., and LOWBURY, E. J. (1950). *J. Hyg., Camb.*, **48**, 6.

(1) Floor dust was collected from the wards of isolation hospitals, houses, and schoolrooms, and the fine dust was separated from the fluff by a sieve, as the former was found to harbour more bacteria. Weighed portions were examined by replicate tests after shaking in fluid with glass beads. The distribution of counts suggested that bacteria were present in aggregates, which were broken down by shaking in this way ; more elaborate methods did not appear to break down the aggregates any more efficiently. The distribution of counts in replicate tests approximated to a log.-normal form and this relation held for pathogens (*Streptococcus pyogenes* and *Staphylococcus aureus*) as well as for non-pathogens.

(2) Floor dust containing *Staphylococcus aureus* and *Streptococcus pyogenes* was exposed for long periods to atmospheres of different humidities. The fall in bacterial population showed a positive correlation with humidity (a possible exception has been found in the case of *Bacterium coli*). The death rate increased more rapidly with relative humidity at high humidities, but there was no maximum death rate at any particular relative humidity.

Bacteria in dust survived longer in a cupboard than in a refrigerator where the relative humidity was between

75 and 90%, the disadvantage of high humidity outweighing the advantage of low temperature.

(3) Although the lethal effect of ultraviolet light on bacteria is much reduced if the bacteria are fixed to dust, it does not follow that such exposure is without effect. Bacteria-laden dust was exposed to ultraviolet light, fluorescent lighting, tungsten lighting, and daylight for long periods. Tungsten light was without effect but the activity of the others was definite though limited, being greater in the first 10 days than thereafter.

In a very dry atmosphere the effect of all the radiations was reduced. At ordinary humidities in rooms, low-intensity ultraviolet light or fluorescent lighting of good intensity destroyed various organisms at a rate five times that experienced in the dark.

The bactericidal effect of ultraviolet light on dried films of *Streptococcus pyogenes* varied with relative humidity and the medium in which the streptococci had been suspended before drying. The death rate was initially logarithmic but frequently slowed down, and surviving organisms seemed to possess a degree of resistance. The death rates of *Strep. pyogenes* in these preparations were greater than those found in floor dust. *Scott Thomson.*

The Persistence of Dust in Occupied Rooms. LOWBURY, E. J. (1950). *J. Hyg., Camb.*, **48**, 1.

As a preliminary study of the survival of bacteria in floor dust, experiments were designed to investigate the persistence of dust in rooms. Small amounts of insoluble fluorescein as a powder or precipitated in blanket wool were scattered in rooms which were thoroughly dusted daily. Measurable quantities were detectable in the floor sweepings for 3 weeks. *Scott Thomson.*

Static Pressure and Ventilation Rates in Rooms. BLACK, F. W. (1950). *J. Hyg., Camb.*, **48**, 44.

GENERAL

Hazards in the Use of Radioactive Static Eliminators and Their Control. SILSON, J. E. (1950). *J. publ. Hlth*, **40**, 943.

Static electricity is created in the course of many industrial processes, particularly where paper, plastics, and rubber are handled ; this can create dangerous risks in industry. For example, in the rubber-coating of fabrics a heavy static discharge develops on the sheet being coated as it passes between spreading rollers. The solvents used in the rubber-coating industry are very inflammable, and a spark might cause a serious fire, or an explosion.

Recently, bars of radioactive substances have been used to ionize the air ; the substances being either polonium or radium. Radium bars have a half-life of about 1,600 years, so that once installed they require no further replacements. Beta radiation is easily controlled by shielding, which can be accomplished by a properly designed housing for the radium bar. Protection against gamma radiation involves the maintenance at all times of a minimum distance between the worker and the source, since shielding is generally impracticable because of the great thickness of lead required. In general, safe working exposure can be obtained by the maintenance of an 18-in. (45-cm.) distance between the worker and the heaviest bar used.

Polonium has a short half-life and produces a hazardous radioactive dust ; this is therefore not so useful. Workers employed in industries where radioactive static eliminators are used should therefore be under constant medical supervision ; film badge monitoring is the only reliable check on the actual dose which they are receiving. *K. M. A. Perry.*

The Carcinogenic Action of Benzidine. SPITZ, S., MAGUIGAN, W. H., and DOBRINER, K. (1950). *Cancer*, **3**, 789.

In an investigation of the origin of bladder tumours among workers in the dyestuff industry, benzidine, some compounds encountered during its manufacture (azoxybenzene, hydrazobenzene, and benzidine sulphate), and some closely related compounds (tolidine, azobenzene, and benzidine disulphonic acid) were tested for carcinogenic activity.

Benzidine was found to be carcinogenic for rats of the Sherman strain, producing carcinomata of sebaceous glands adjacent to the auditory canal, hepatomata, and adenocarcinomata of the rectum. Bladder tumours were not produced. Tumour formation was caused both by technical-quality and specially purified benzidine. Attempts to influence the carcinogenic action by variation of the diet gave inconclusive results. Benzidine sulphate, administered orally and parenterally to rats, had a similar but less potent carcinogenic activity. No tumours were obtained with any of the other compounds tested.

No tumours occurred in rabbits or monkeys after injection of benzidine. In 1 out of 7 dogs fed with benzidine for 5 years and surviving for from 5 to 8 years, a papillary carcinoma of the urinary bladder was found on cystoscopy, but no conclusions about the carcinogenicity of benzidine for dogs can be drawn from this single occurrence. *L. A. Elson.*

The Industrial Aspects of Cardiac Infarction. STRONG, G. F. (1950). *Ann. intern. Med.*, **33**, 690.

An account is given of 20 patients allowed compensation in British Columbia for cardiac infarction as an industrial disease.

In the first case a 47-year-old policeman ran 2½ miles in pursuit of a burglar, then drove a car at rapid speed and under considerable tension for about 5 miles, and finally collapsed.

It is suggested that in industrial workers cardiac infarction should be compensated when it occurs after penetrating or non-penetrating wounds of the chest, when it occurs during shock following accidents or the operations necessitated by accidents, and when it follows burns or asphyxia, or extra physical effort resulting in physical strain. On the other hand, when it occurs during working hours without any antecedent factors it

would hardly warrant the serious consideration of compensation boards.

It is evident that if cardiac infarction is to be regarded as an industrial accident the time relation is of great importance. The cardiac symptoms should appear at the time of the effort and be more or less continuous until the diagnosis of infarction is established. Of the 20 patients discussed, 8 have died, and of the 12 surviving 9 have some continuing disability, 2 have returned to work, and one is still convalescing. The result of compensation is that the patients are imbued with the idea that their heart condition developed as a result of exertion and that any future activity will be harmful. This, coupled with the comfort of permanent disability allowance, means that complete recovery is almost unknown.

Rehabilitation of individuals with coronary sclerosis is of the greatest importance ; industry must be prepared to keep these individuals in jobs suited to their cardiac capabilities. On the other hand, industry must not be penalized if one of these succumbs to a cardiac infarction following a minor accident or strain insufficient to cause death in a healthy individual. *K. M. A. Perry.*

Protective Clothing for Work with Mercury and its Cleansing. MIGAJ, K. V. (1950). *Gigiena*, No. 6, 17.

Experiments were made to determine the best type of clothing for use as a protective against mercury vapours or particles. Sundry textile materials were used ; calico took up a smaller amount of mercury than any other material tested. The greatest adsorption occurred with army-uniform cloth. The tests were all made on the laboratory bench, and mercury in the material was estimated colorimetrically. For removal of mercury, washing was most effective if two changes of soft water were used, this technique being superior to any physical or chemical method tried. If exposed to the air, most material became nearly or completely free of mercury after 4 days. *G. C. Pether.*